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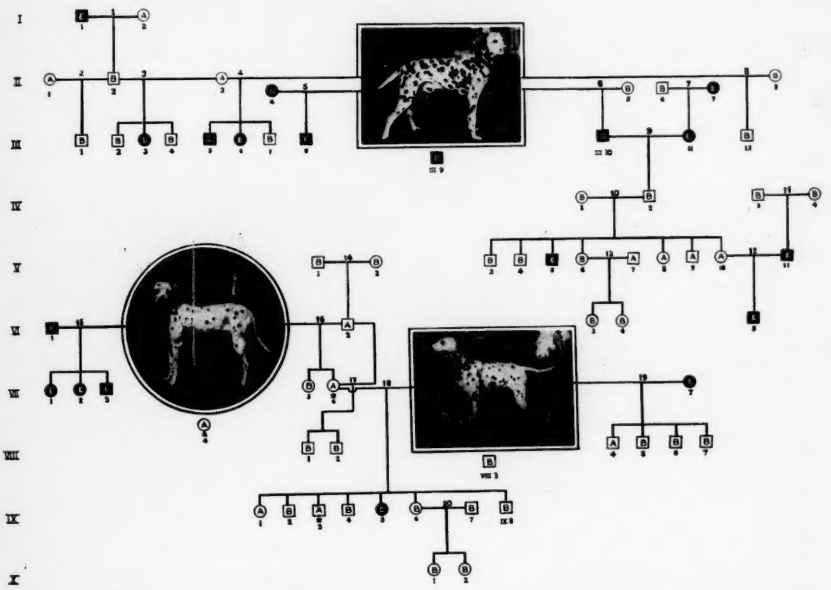

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Genetics of Nervous Disturbances
Deformed Arms Inherited

VOLUME 31 FEBRUARY, 1940 NUMBER 2

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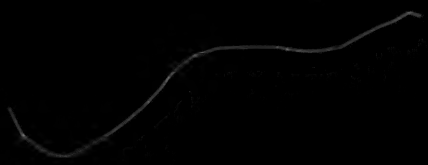

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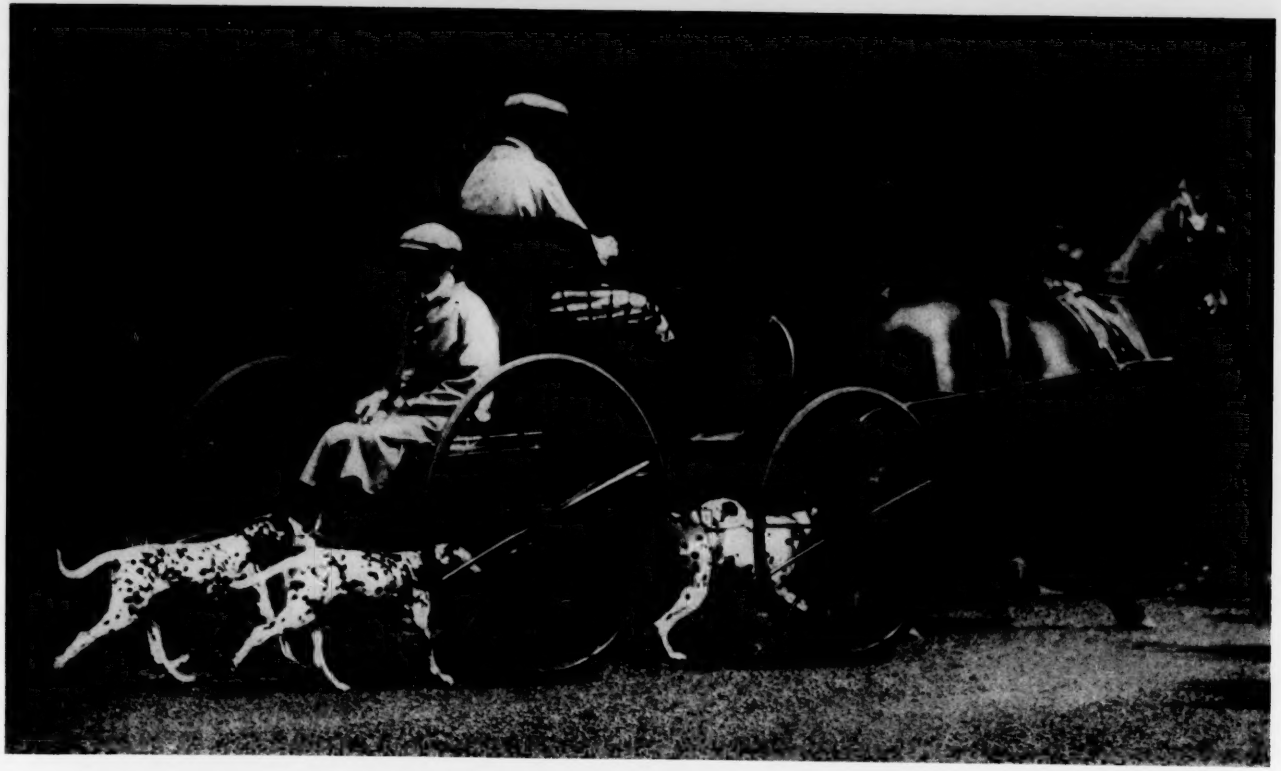


TRAINING THE DALMATIAN COACH DOG TO "COACH"

Frontispiece

Man uses a variety of inherited behavior patterns in developing his domestic animals. The peculiar position of the Setter and the fetching instinct of the Retriever both being trained hereditary patterns. The behavior of the Dalmatian coach hound, a breed which instinctively prefers horses to men, is also of this nature. The inborn instinct of the dogs is fixed by training, but the positions in which the dogs run, under or behind the coach, appear to be more a matter of heredity than of environmental training. This photograph shows a "Dalmatian training school" literally in progress, with trained older dogs leashed to the youngsters going to school. The forward position under the coach is the preferred position for the coach dog to occupy, but not all of them will adopt it even if forced to run there during the training period. The dogs shown here are: at rear, Romance and Wigwag; at front, Gideon and Gideon's son, shown in the pedigree chart (Figure 2 as III-10, III-9, IX-8 and VIII-3).

INHERITANCE OF POSITION PREFERENCE IN COACH DOGS

CLYDE E. KEELER AND HARRY C. TRIMBLE
Wistar Institute of Anatomy and Biology, and Department of Biological Chemistry, Harvard University

IT is often difficult to draw a line between instinctive behavior and an inborn ability to learn. An untrained Setter pup romping in the back yard, may suddenly freeze and point to a robin on the fence, holding his point with the silence and discipline of a champion. Such a reaction in a state of nature would not be to the Setter's advantage, but the sportsman seizes upon this instinctive reaction and trains it for his own purposes, carefully selecting those animals best endowed with the ability to make this unusual response.

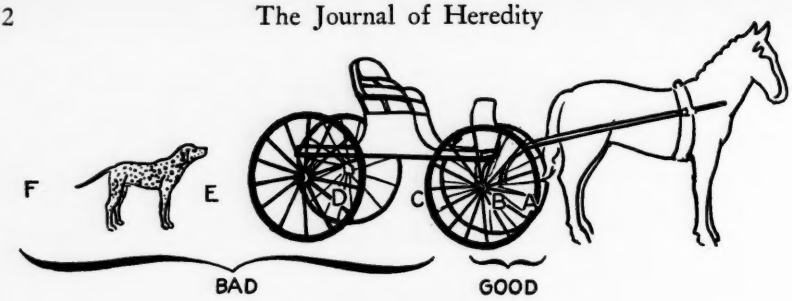
A few western cats and practically all Siamese cats are natural-born retrievers although because of their independence it is often difficult to teach them other tricks. If we had a use for this ability it could be trained. The retrieving trait is dominant in crosses and in one hybrid kitten raised by the senior author, the performance was repeated eighty-three times in succession until the animal dropped from exhaustion.

To a given situation certain individual animals make instinctive reactions quite abnormal for their species, reactions that may serve no useful purpose. Thus, a certain family of inbred Wooler rabbits instead of immediately stomping and then running away when frightened, rise up quickly on their hind legs and jump straight into the air. If man found a use for this reaction he could undoubtedly select it as he has the reactions of Pointers and Setters. Certain shaker mice when excited turn backward somersaults instead of running away. Many other such bizarre inherited tendencies capable of training could be cited in our domestic animals.

For more than a century on English and American estates the highly social Dalmatian Coach Hound has been selected for its desire and ability to run under a wagon or carriage as shown in the Frontispiece. Some persons rearing and training this breed say that they never had a Dalmatian that did not "take to coaching," but this statement may be considered a professional exaggeration, although it seems certain that a majority of Dalmatians actually do. That this reaction is not all training, however, is evidenced by the fact that an untrained animal of able stock may occasionally break from his leash when a carriage goes by, not to chase the horses, but to run happily under the carriage where he feels instinctively that he belongs. This instinctive reaction may frequently take place in six-months-old dogs but occasionally does so in much younger individuals. A very serious puppy of two and a half months is known to have felt the coaching urge so strongly that he forsook his playmates each day while a lawn was being graded in order to paddle solemnly along under the wheelbarrow. Some breeders aver that the secret is the Dalmatian's inordinate love of horses that makes them want to follow closely at their heels, not only in coaching but also when in saddle, and that this hippophilic characteristic is expressed by their desire to be with horses in their stalls, which situations have no appeal to other canine breeds. Many Dalmatians, if given an opportunity to choose whether they want to live at the house with men or at the stable with horses, prefer horses.

Yet merely the tendency to run under a vehicle or to follow a horse is not the only characteristic of this mental trait which is inherited. The data to be presented show that Dalmatians differ individually with respect to the precise position which they take in running under a carriage when a choice is permitted.

dogs shown here are: at rear, Romance and Wigwag; at front, Gideon and Gideon's son, shown in the pedigree chart (Figure 2 as III-10, III-9, IX-8 and VIII-3).



"COACHING" POSITIONS

Figure 1

Coach hounds run customarily in various positions, from just back of the horses' hind legs to considerably in the rear of the coach. The farther back the worse the position. Every coach dog has a definite preference as to the position he takes, and this is not changed by training.

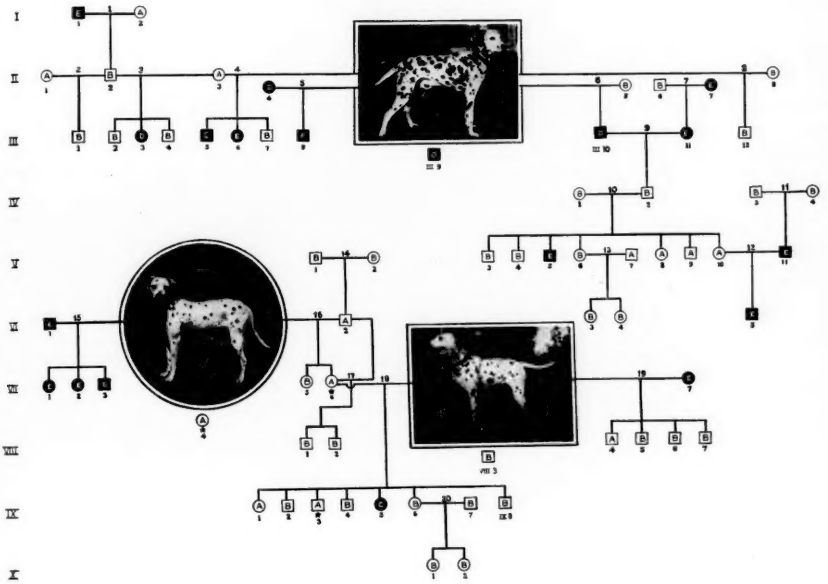
One coach dog enjoyed the front axle position when a team was driven but when the old coach-and-four lumbered out of the stable he always took a precarious position under the pole, completely boxed by the two lead and the two pole horses. At turns in the road he left his box through the open side, cautiously slipping back into it again when the train had straightened out. Once in a certain parade a band immediately preceded the coach. This extremely forward dog forgot himself and marched behind the band in front of the lead horses. Other Dalmatians with the coach in the same parade stuck to their normal positions.

One rarely has an opportunity to study the variations in coaching ability, because most persons who have used Dalmatians for coaching try them out several times alongside an experienced dog, and if they do not come up to standard discard them or at least eliminate them from their breeding rosters. We have examined carriage position in a group of Dalmatians which were not always discarded as breeders because of lack of coaching ability. Our data show that something of this preference for particular coaching positions may be transmitted to offspring.

In a large kennel of Dalmatians which we have been permitted to study, it has been the practice for more than 25 years to train the animals to follow horses and run under carriages. This training usually began shortly after the dogs were six months old. In the beginning a pup would be taken out with its collar fastened to that of a trained dog and the pair led behind the carriage at gradually increased rates of speed. In most instances the leash could be omitted in a short time and the neophytes then were permitted to seek the particular position for running which suited them best. It has been observed throughout this long period that dogs did have individual preferences for particular positions and that they always sought those same places. We have designated the different positions with reference to the

TABLE I. Rating of Offspring

	Class	Mating	A	B	C	D	E	F
1		II		1				
2	I			1				
3	I			2		1		
4		II		1	1		1	
5		III						1
6		II				1		
7		II					1	
8		II		1				
9		III		1				
10	I		3	3			1	
11		II					1	
12		II					1	
13	I			2				
14	I		1					
15		II					3	
16	I		1	1				
17	I			2				
18	I		2	4	1			
19		II	1	3				
20	I			2				
		Totals	8	24	2	2	8	1



PEDIGREES OF COACHING ABILITY

Figure 2

In most coach hound breeding, only dogs which run in preferred positions (*A* and *B*) are used. This series of pedigrees is from a kennel in which such selection was ignored in favor of the excellent coat pattern of III-9, an "unconventional" dog who ran in the *D* position under the rear axle (see Figure 1). The black symbols show dogs who instinctively occupied bad positions.

horses and carriage in accordance with the letters shown in Figure 1. The preferences of the animals studied are presented in Table I.* In this table it can be seen that dogs of the Dalmatian breed have definite differences with respect to the eagerness with which they follow horses and carriages. Since approximately 70 per cent of the animals tested chose those positions which entitled them to be rated as "good" coaching dogs, it is evident that this trait is well intrenched in the breed.

Data with respect to the inheritance of these preferences are presented in Figure 2. From these the following are typical observations. A certain bitch was accustomed to run with her head in a position to touch the horse's leg. She was mated (mating 1 in Figure 2) to a dog which always trailed the carriage. The three offspring (2 females and 1 male) trailed the carriage so badly that their line was discontinued. The same bitch was mated to a dog that ran with his head far forward of the front axle (mating 16). This pair had two female pups, both of which ran very far forward, one even keeping her head between the horse's hind legs. This extreme forward-running bitch was mated to her father (mating 17). She produced two male pups, both of which preferred the forward position. She was then mated to a dog preferring a far forward position (mating 18). They produced a

*The data upon which the table is based were given to us by the owner, the kennel master, and the trainer at different times. In every instance these three people were in complete agreement with respect to the behavior of individual animals.

litter of seven pups; six of these preferred the forward places and one insisted upon runing with his nose touching the horse's hind leg. The far forward-running male parent was mated (mating 19) to a female whose coaching score was very "bad". This pair had four offspring; all preferred the forward position.

In another strain a particular male preferred the back axle. Because of his splendid markings he was mated to four females (matings 4, 5, 6, and 8). Six of his offspring were tested for coach running. Three are known to have been "bad". One of these ran under the rear axle, one trailed behind the carriage, and one would not follow horses or carriages at all.

From the foregoing data it is evident that something which determines the preference of position in carriage running may be inherited. For comparison of this character we have assigned alphabetical ratings (see Figure 2) to those animals for which information on their coach running characteristics is available. Based on these ratings of the parents the different matings have been classified as class I (good × good), class II (good × bad) and class III (bad × bad). Thus we found that there were nine class I matings (nos. 2, 3, 10, 13, 14, 16, 17, 18, 20) which produced a total of 7 *A*, 17 *B*, 1 *C*, 1 *D* and 1 *E* offspring; there were nine class II matings (nos. 1, 4, 6, 7, 8, 11, 12, 15, 19) which produced a total of 1 *A*, 6 *B*, 1 *C*, 1 *D*, and 7 *E*; there were two class III matings which produced a position *F* pup and a position *B* pup.

If we score the positions *A, B, C, D, E, F* as 1, 2, 3, 4, 5, 6 respectively (although squaring the distance might be mathematically more sound) it follows that class I matings produced 24 good dogs with an average rating of 1.71 plus three bad dogs with an average of 4.00. Class II matings produced seven good dogs with an average rating of 1.86 plus nine bad dogs with a rating of 4.66. Class III matings produced two pups with an average rating of 4.

Not only is there a greater percentage (53% : 11%) of bad dogs from class II matings than from class I matings, but the average grade of good dogs from class II matings (1*A* : 7*B*) is inferior to those from class I matings (8*A* : 16*B*). The bad dogs from class II matings average worse than the bad dogs from class I matings. Only one individual out of three bad dogs from class I matings had as bad a rating as 5, whereas among the bad dogs from class II matings seven out of eight rated as badly as 5.

Because the ability seems to be reduced all along the line by bad matings, it seems fair to compare the total scores for offspring of the three types of matings. The 27 progeny of No. I matings made an average score of 3.23; the sixteen descendants from No. II matings made an average score of 3.44. The two offspring of No. III matings made an average score of 4, as noted above

Because of valuable show bench characteristics that they possessed, certain dogs "bad" from the coaching standpoint were used as breeders in several crosses to "good" dogs. In view of the results of matings Nos. 5 and 15 and on account of the experiences of other breeders, class III matings of "bad" × "bad" dogs were not continued long and mixed matings became relatively few. From a statistical standpoint this omission is unfortunate for our studies, but even the restricted results of the two "bad" × "bad" matings (Nos. 5 and 9) gave worse scores than those resulting from "good" × "good" matings.

Two of the dogs in the colony which were classed as failures from the coaching standpoint were also described as "man-shy". This description suggests the possibility of some relationship between natural timidity and poor coaching ability.

Conclusions

1. Dalmatian dogs trained for running beneath carriages have individual preferences for distance behind the horses.

2. Something connected with these preferences appears to be inherited.

3. It is possible that "bad" coach following may be an expression of general timidity.

PROBLEMS SET BY CLINICAL NEUROPATHOLOGY TO BE SOLVED BY GENETICS

S. DAVIDENKOV
Leningrad, U. S. S. R.

IN OBSERVING inherited diseases the clinician is invariably faced by a number of questions which he himself for the most part fails to solve. My intention is to touch upon some of them which especially refer to affections of the nervous system, but I know that similar questions must be troubling the minds of physicians in other branches of medicine as well.

I. The Problem of Duplex Inheritance

A large number of inherited nervous affections are known to exist, which behave both as recessives and as dominants. Clinically the two forms are either barely distinguishable from one another (as in the case of spastic paraplegia) or are somewhat different, giving cause in the latter instance for vast discussions in the medical press, as to whether they are independent diseases or merely variants of the same affection (Friedreich's recessive ataxia and Pierre-Marie's dominant ataxia is an example). Sometimes one of the two variants is of far more frequent occurrence than the other. Thus, in the vast majority of the cases Charcot-Marie's amyotrophy follows the dominant type, while the recessive is exceptional (Steinthalt's observation and a case observed by us). The same statement is equally true with respect to myopathy, while in cases of hepatolenticular degeneration the relation is converse. This mimicry in clinical forms is of so common an occurrence, that it seems to demand a uniform explanation, which so far evades us. The question is whether the phenomenon is due to the formation of multiple allels or to position-effect, or again to the accumulation in the family background of modifiers affecting the degree of dominance possessed by the gene (R. Goldschmidt's[4] "dominigenes"). The latter assumption, which appears to be very probable, would have required the distinction between the dominant and the recessive forms to have been far less marked, and the occurrence of a number of intermediary cases, viz., of irregular dominant forms, of those characterized by low penetrability, etc. This, apparently, is practically what is found to occur in the case of Charcot-Marie's amyotrophy. The problem here touched upon as yet finds but few analogues in experimental genetics, which may partly be due to the fact that it has failed heretofore to be clearly enough formulated.

II. The Problem of Translocation

This question has been raised by R. C. Punnet[9] in connection with the phenomenon frequently observed in hereditary opthalmology, where similar diseases are inherited in some families as autosomal dominants, and in others behave as sex-linked recessives. A few instances of the same kind might be found to occur in the clinic for nervous diseases. To confirm the correctness of the translocation hypothesis an attempt should be made to find critical pedigress which would show one type of heredity being converted into the other. Barigozzi[1] has recently published one such pedigree in connection with hereditary nystagmus, but the family in question has too few members, so that Herringham's[5] earlier observation in connection with Charcot-Marie's amyotrophy is far more complete. In the latter family the recessive sex linked type of heredity is very pure; yet one male had a son who

was affected. The question arises whether this might not have been due to the non-disjunction of his wife's X-chromosomes. The problem advanced by Punnet has not been so far satisfactorily solved, but this is more the fault of the clinicians themselves who have failed to collect sufficiently convincing pedigrees, than of the geneticists who have studied the problem in very great detail.

III. Penetrance

Contrary to this it is the clinician who insists on taking up the study of *the effect produced by environment on the penetrability of genes possessing a low power of manifestation.* The effect can clearly be seen in studying certain clinical forms. By way of an example consider Erb's type of myopathy which follows the autosomal dominant type of inheritance but is usually a highly inhibited form. Occasionally in one generation highly developed forms suddenly make their appearance. Physicians who are concerned with genetics have always found a difficulty in interpreting this kind of pedigree. Having studied a number of similar pedigrees in our clinic we have been led to draw a somewhat unexpected conclusion: the percentage of heavy drinkers among the fathers of our patients was excessively high, i.e., out of the 26 fathers of the subjects affected with myopathy whom we observed eleven suffered from alcoholism and of those nine in a very severe form. Thus, the percentage considerably exceeded the frequency of alcoholism observed in the control group.[6] This seemed to suggest that the phenomenon was due to peculiar blastophtoria which increased the capacity of the chromosomal factor for manifesting itself. Apparently, the role of congenital syphilis in the origin of the so-called syphilitic Friedreich's symptom-complex is practically the same. Yet as far as my knowledge of special genetical literature goes I have failed to find that sufficient attention has been given by experimenters to this problem which is of great importance to the clinician.

IV. Heterozygosity

Further, a peculiar and purely genetical problem arises from the consideration of the numerous facts observed in the neurological clinic as to the *correlation existing between symptoms depending on the hetero- or homozygous structure of the same gene.* Leaving aside cases in which the difference between these symptoms is of a purely quantitative nature, it is of particular interest to determine whether or not a symptom not particularly characteristic of a homozygote can be characteristic of a heterozygote. The relation existing between migraine and genuine epilepsy might serve as an illustration. The former is highly typical—particularly in women, of the heterozygous structure of the recessive or even intermediary gene of epilepsy. It is true that female epileptics sometimes also suffer from migraine but far less frequently than their heterozygous female relatives. Every neuropathologist has had occasion to observe the curious combination of a female epileptic who is free of migraine and whose mother was a victim to severe migraine; the father in these cases usually shows signs of either epileptoid psychopathy or alcoholism. Yet migraine is by no means the same disease as epilepsy. The determination of the relation existing between the hetero- and the homozygous characters would greatly help the physician to understand this kind of phenomenon.

V. Gene Interrelation

The next question refers to those *minor dysplasiae* which the neuropathologist has frequent occasion to observe. In considering them one is led to believe that the same dysplasia often occurs in different forms: sometimes it is mildly manifest, often variable and frequently irregularly inherited. At other times it is markedly manifest and clearly Mendelian. We have observed families with mild hereditary anomalies of the sternum; and parallel to them families affected with developed koilosternia or "hollow chest," as observed by Snyder and Curtis.[10] We have also observed

families with minor and variable, though always definitely hereditary curvatures of the fingers; and parallel to them families in whose members the inheritance of hook-like little fingers regularly follows Mendel's law. The question is whether the genes which lie at the bottom of these similar dysplasiae are of an entirely independent nature or are in some way connected with each other possibly as multiple allelomorphs or linked modifiers. All this is by no means clear. Yet, it has to be admitted that it is from the anthropologist rather than the geneticist that the physician should expect to find an answer to questions of this kind.

Among the above unsolved problems there is a particular problem which has been especially studied in our clinic within the past few years, viz.: *the inter-relation existing between genes having a similarly directed effect.* Experimental genetics has at its disposal but a small number of experiments of this type, but clinical observations emphasize that further investigations should be continued precisely in this direction.

VI. Mildly Affected Relatives

Our speculations have been based on two kinds of facts:

In the first place many affections of the nervous system are found to be attended by an abundance of dysplasiae which also frequently occur in relatives of the patients who are seemingly neurologically sound. The frequency of dysplasiae in the relatives has been found to be far greater than might have been expected. This could perhaps be regarded as an indication of the presence in an inhibited form of the same gene that determined the developed dysplasiae. Similar dysplasiae are polymorphous, but by no means specific so that they exclude the possibility of differential diagnosis. For instance: among the relatives of patients affected with Friedreich's ataxia we not infrequently find people who are perfectly sound in every other respect and yet have pedis excavati, in a very mild form, or some anomaly of the tendon reflexes. At the same time anomalies which are phenotypically indistinguishable from the former are found to occur in the relatives of patients suffering from either Charcot-Marie's amyotrophy or syringomyelia, not to mention that a large number of excavated feet and cases of congenital weakness of tendon reflexes are observed to occur among the population outside any connection with family ataxia, Charcot-Marie's amyotrophy or any other diseases. Similarly a number of other anomalies which play an important part in the clinical picture of many nervous diseases can be very frequently observed in a milder form in perfectly sound people.[3] Among these latter anomalies we shall mention pes planus, clinodactylia, camptodactylia, "hollow chest," anomalies of the spine, enlarged head of the ulna, shortness of the peroneal portion of the foot, congenital variations in the degree of development of intraneural connective tissue (the so-called "thick nerves"), shortness of muscular bodies together with incongruously long tendons, etc. Moreover, many of these anomalies are characterized by a fairly regular hereditary transmission. Consideration of the above data has led us to assume that these numerous micro-anomalies mendelize in the population in general owing to certain independent inherited factors, apparently, of a multiple nature, but that the genes which cause certain nervous affections, such as Friedreich's disease, Charcot-Marie's disease, Strümpell's disease, hypertrophic neuritis and syringomyelia—have the capacity for *increasing to a more manifest degree certain micro-anomalies if they occur in a given genotype.* Apparently, it is precisely this dissimilarity of the original genotype which accounts for certain clinical differences observed in separate cases. The pleiotropism of certain pathological genes should in this case be regarded as being to a great extent conditional. For instance: Friedreich's ataxia is usually attended by a characteristic deformation of the foot but this is found to occur in precisely those cases in which the family background also shows a tendency

towards deformities of the foot. In some cases, however, Friedreich's disease is not characterized by any deformation of the foot; the examination of the patient's relatives in one of the latter cases revealed the complete absence of any micro-anomalies in the structure of either the foot or the toes. In another such instance Charcot-Marie's muscular atrophy is not attended by hypertrophy of the nerves if its gene is found to split in a family which is free of the "thick-nerve" micro-anomaly, i.e., in which the somewhat excessive development of the intraneural connective tissue still remains within the physiological limits. Contrary to this, a manifest clinical picture of "hypertrophical neuritis," which some neuropathologists are apt to regard as an independent clinical form, is found to develop if the "thick-nerve" micro-anomaly is present in the family. Again, as a rule Friedreich's disease is not attended by ptosis, yet in one case we found that the subject under observation had markedly developed ptosis of both the upper eyelids. It turned out that many of his relatives had the physiological characteristic of "lowered lids" and the hereditary course of this latter feature definitely failed to coincide with that of Friedreich's gene. In comparing numerous data of a similar kind it has been found that the connection existing between our major pathologic genes and the micro-anomalies is of a highly specific nature. Thus, Charcot-Marie's amyotrophy is very sensitive to the "thick-nerve" anomaly, while Friedreich's ataxia which greatly resembles the former disease in many respects remains indifferent to the same micro-anomaly. Contrary to this, Friedreich's ataxia markedly intensifies the family tendency towards deformations of the spine and mental backwardness. Along with this, it has to be admitted that many of the forms share in certain respects a similar conditionally intensifying effect, a fact which, apparently, explains the resemblance of their outward manifestation in their inhibited or rudimentary forms. My collaborators and I have collected a number of clinical facts which serve to confirm the above statements.[2] These new points of view have also led us to draw certain important practical conclusions. To begin with, it has proved to be impossible to follow our previous practice of ascribing all the minor anomalies occurring in the patient's family, and reproducing in miniature those observed in him, to the one basic gene; for some of these micro-anomalies may be in no way connected with the basic gene. It is highly probable that in some of the cases at least it is not because of the patient's having these characteristics that they are found to occur in the family, but conversely, the fact that there is a corresponding family background determines the patient's status. It clearly follows how greatly this complicates the diagnosis of the heterozygous or inhibited forms of the disease or, rather, how it may help us to avoid the error which must have been very frequent until the question of conditionally intensifying pleiotropism of our "major" neurotropic genes was raised.

VII. Other Perplexities

Other facts we have been studying lately are as follows:

It has long since been mentioned in special literature that the most various nervous and psychic anomalies preferably combine in the same individuals or the same families. Much has been said on "neuropathic families," "degeneration," etc. The genetical mechanism of this attraction of heterogeneous diseases to one another was incomprehensible and demanded a more detailed study. In our clinic we have failed to find any confirmation of some of the statements in the literature concerning the frequency of combined forms. For instance, the number of individuals affected with epilepsy in families having syringomyelia and myopathy very nearly coincided with the number of epileptic cases as observed in sound families. ($0.64 \pm 0.28\%$., $0.61 \pm 0.26\%$. and $0.62 \pm 0.35\%$.). An erroneous conclusion had apparently been drawn owing to the fact that only the selected

combined cases were taken into account instead of all the cases observed. However, though it has proved impossible to support the conception of a "general" neuro-psychic encumbrance, there are separate, strictly definite forms which indeed are frequently found to occur jointly in the same family. Some of these forms are genetically connected with one another and in this case their alternating within the same family can readily be explained (for example, migraine and epilepsy). Other forms are *a combination of genes which are known to be of different natures.* Why, then, are they found to manifest themselves jointly? This is a very complex problem which most certainly cannot be solved by the notion of "affinity," which fails to explain anything. In trying to find a solution of this problem we have once more found it possible to follow the same train of thought as before, i.e., to take into account the possibility of the *intensifying effect of the summarized heterogeneous neurotropic genes.*

There exists in the clinic of nervous diseases no small number of genes possessing low penetrability or a highly variable expressiveness which cause the appearance of developed cases in turn with more or less inhibited or rudimentary forms. We shall cite, as an instance, atrophic myotonia, leukodystrophia and Gélineau's narcolepsy. Rudimentary conditions often make it possible in these cases to trace definitely the course run by the hereditary factor. These include an isolated cataract or separate myotonic or atrophic symptoms in families affected with atrophic myotonia; mild spastic paraparesis or other neurologic symptoms occurring in families having progressive leukodystrophia, and more or less mild anomalies of sleep in families of narcoleptics.

We have succeeded in finding in some of the pedigrees referring to above cases, which were sufficiently complete to give us a chance of taking into account *marriages that preceded the appearance of developed forms.* These data showed that the carriers of the dominant morbid gene always had in these cases for partners individuals suffering from either epilepsy, or some other mental disease. The regularity of this occurrence was particularly evident in Gélineau's narcolepsy. In some of the families it was sufficient to join the epileptic gene in its heterozygous structure to cause the appearance of sick children. In other families—in which the penetrability of the narcoleptic gene was apparently lower—cases of manifest narcolepsy were found to occur only providing there was a chance of homozygosity on the part of the epileptic gene. This naturally led to the assumption that in the latter case we had to deal with the effect produced by such nervous anomalies as epilepsy, schizophrenia or certain massive psychopathies on the degree of penetrability of such incompletely manifest forms as atrophic myotonia, leukodystrophia or narcolepsy. The assumption helped to explain why in these families patients often revealed simultaneously various other neuropsychic anomalies. Sometimes it proved possible to trace how these affections no longer merely intensified the penetrability of a certain pathologic gene, but its expressivity as well. The connection between Ménière's disease and migraine or epilepsy may serve to illustrate the situation. The connection has often been pointed out and indeed has been fully confirmed. However, it was very difficult to explain this connection, as congenital vestibular hyperaesthesia, on the ground of which Ménière's disease mostly develops, depends on the readily traced dominant factor, which is partially limited by the female sex, whereas epilepsy and its milder heterozygous manifestation, migraine, depends on a special recessive factor. It seemed inexplicable why these two different genes should appear so frequently together. The analysis of these families revealed, however, an interesting fact. Namely, that Ménière's syndrome ran a far more severe clinical course in families which at the same time suffered from migraine or epilepsy.[7] Since it is always the severest cases who consult the physician the more severely affected subjects had uninten-

tially been selected for observation, i. e., individuals whose families prove to be most heavily encumbered with the epileptic gene.

N. V. Borovka has made an interesting mathematical analysis of a case in which the basic dominant gene did not possess a complete degree of penetrability, while its modifier which is in no way connected with it is capable of increasing its penetrability to a greater or lesser degree. (It has been assumed that a homo- or a heterozygous structure of the modifier changes the penetrability of the basic gene to an equal extent.) The carriers of the former character in this combination must necessarily have among them a larger number of carriers of the latter character than the average population. Not being able to discuss this investigation in detail, I shall just mention that the degree of this increase, χ, depends on how far the second gene intensifies the penetrability of the first one (w) as well as on the concentration of the second gene ($K_2 = \frac{g_2}{p_2}$), χ being the higher, the lower K_2, according to the following formula:

$$\chi = 1 + \frac{W - 1}{1 + W\ (2\ K_2 + K_2^2)}$$

This can be illustrated by a concrete example: when $W = 10$ and $K_2 = 0.01$, χ will be equal to 8.5 for the propositi, i.e., we have reason to expect to find carriers of the modifying gene among carriers of the primary character 8.5 times more frequently than among the average population. Therein probably lies one of the possible explanations of the relative frequency with which certain genetically independent nervous diseases appear jointly.

The clinician who does not know what is happening to the chromosomal apparatus of his patients is forced to confine his attention to what he can see occurring "on the surface" of these processes. But on the grounds of this study, which is necessarily limited by phenotypes and geneologies he may form certain generalizations, often of a hypothetical nature. Naturally he desires his conclusions to be confirmed by experimental genetics. Has it been possible to observe experimentally the summation of the pathologic effect produced by neurotropic genes? As far as my knowledge of genetic literature goes this problem has thus far been treated only by single experimenters.[8] This may be due to the fact that a purely theoretical investigator fails to see the practical actuality of this problem, while for the clinician the problems here touched upon are of paramount importance. To draw greater attention to them has been the object of the present communication.

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Tetraploid Pears

A bud sport from the "Fertility" pear ($2n = 34$) has occurred in Messrs. Seabrook's Nurseries. This new form, called "Improved Fertility," exhibits increased size in all its parts. The average weight of each fruit is 197.5 gm. compared with 125 gm. in the original form. M. B Crane and P. T. Thomas (*J. Genetics*, 37, 2287-300; 1939) have analysed the new form and found it to be a tetraploid $2n = 68$. This "Improved Fertility" is more self-fertile than the original diploid form and may give rise to further desirable tetraploid varieties of pears. The diploid form, although known in commerce for sixty years, is comparatively unstable and sometimes shows aberrant cell divisions.—*Nature*, April 1, 1939.

CHEMICAL INDUCTION OF GENETIC CHANGES IN ASPERGILLI

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THE discovery of the ability of nutrient solutions containing mannitol and sodium nitrite to cause the formation of mutants in the fungi, *Aspergillus niger* and *A. amstelodami*, has been recently reported by the writers.[5] The types of genetic changes described could all be classified as "injury" mutants. They exhibited in varying degree and combination differences in loss of ability to undergo complete normal differentiation. They duplicate forms occasionally found in nature. In view of the well-known action of nitrous acid on amino nitrogen it was postulated that the genetic changes brought about were effected through the stripping of amino groups from the proteins concerned in the transmission of hereditary characters in these organisms.

It has seemed desirable to obtain evidence on several points. Extension of these results to other species of *Aspergilli* seemed advisable to determine whether they also displayed a similar type of mutation. A measure of the frequency with which mutations could be brought about might be helpful. If the assumption that nitrites act through the destruction of amino nitrogen groups is correct it seemed probable that other chemical compounds destructive to the amino group should behave similarly. Lastly, other compounds might be found having a similar property to that of nitrite which, alone or in combination with nitrite, might lead to other types of mutation.

A group of species of *Aspergilli* whose mutants might be helpful as an aid in the classification of these forms was selected for the first series of tests. They are listed in Table I, together with data on the appearance of certain extreme types of mutants obtained. The conidia and sclerotia are asexual reproductive bodies, whereas the perithecia contain ascospores produced by a sexual process. Sclerotia are usually considered to be aborted perithecia. It will be noted that the mutants obtained were quite of the same type previously reported for *Aspergillus niger*. That is, they are "injury" mutants exhibiting a loss to various degrees of the power to differentiate. As in the case of *A. niger*, it was not unusual for the same, or closely like, forms to appear in duplicate flasks or in successive experiments.

Some of the mutants obtained deserve special mention. Three types of sterile mutants were obtained with *A. fumigatus:* one with long aerial hyphae completely filling the tube, another with short aerial hyphae, and the last practically free of aerial hyphae, tumulose, and somewhat darker in color. The last type mentioned showed a tendency to transform into the second type; *A. niger* produced a like form that also tended to change similarly. An asexual mutant obtained from *A. fischeri* had conidia formation so greatly enhanced as to insure classification as a form of *A. fumigatus*, were its origin unknown. Though the strain of *A. variecolor* studied does not form a continuous growth on the surface of Czapek agar slants, one of the mutants obtained did do so. Perithecia formation could not be completely suppressed in this species, nor in *A. amstelodami* though continued work might have resulted in a completely asexual form; or a higher concentration of nitrite might be necessary.

All mutants induced by nitrite treatment exhibited a decrease in sexual reproduction. In some instances perithecia were completely suppressed. Sclerotia were similarly affected in the mutants. The similarity in behavior of perithecia and sclerotia to nitrite treatment of the

organism would appear to indicate a close morphological and physiological correspondence between these structures. Formation of conidia was not influenced in a uniform direction. Many forms were obtained that were completely sterile. Other variants, however, as with *A. amstelodami* and *A. fischeri,* displayed a greatly increased ability to form conidia as an accompaniment to diminished perithecia formation. When present, the characteristics of conidia and ascospores in the mutants were identical with those of the standard cultures.

Other experiments with *A. niger* were undertaken in an effort to render clearer the chemistry of mutant induction. The effect of the addition of other compounds when supplementing the action of nitrite was studied in one trial. The compounds used included colchicine in amounts from 20 to 5000 mg. per liter. Tested at 50 mg. per liter were acenaphthene, acridine, acridine yellow, acridone, aniline blue, methylene blue, methylene violet, phenanthrene, methylcholanthrene, 1, 2, 5, 6 — dibenzanthracene, trioxymethylene, and hexamethylenetetramine. Malachite green was used at 1.0 mg. per liter. The genetic variants formed were quite similar to those formed by nitrite treatment alone, except that four of the forms isolated had the reverse of the mycelial felts of a pink or reddish color.

Fourteen of the twenty flasks in this experiment gave indications of the presence of variants. The changed forms could not be isolated in two instances, while the other twelve flasks gave a total of fifteen isolations. Many of the changed forms were quite similar in appearance. Under the proper conditions, therefore, it may be conservatively estimated that at least fifty per cent of the trials undertaken with nitrite treatment should produce mutants. A repetition of this experiment with one-tenth the quantity of nitrite failed to produce a single variant.

The constitution of the carbon source would seem to influence though not determine the production of mutants with nitrite. In one comparison eighty per cent of the cultures formed mutants in the standard manitol-nitrite solution, whereas a similar sucrose-nitrite solution gave only forty per cent. The types of genetic variants formed were quite similar in appearance with both mannitol and sucrose.

In order to obtain additional evidence on the mode of action of nitrite, the action was investigated of compounds other than nitrite that are known to be capable of eliminating amino nitrogen from amino acids and proteins. These included ninhydrin, chloramine *T,* and potassium iodide. Also included were potassium bichromate,[3] the sodium salt of salicylanilide,[1] and formin because of its property of forming formaldehyde when acidified. All six compounds selected behave similarly to sodium nitrite in that they are relatively stable in neutral solution but become reactive under the influence of acids formed by the organism.

These tests also were limited to *Aspergillus niger* and included a comparison of the effects of sucrose and mannitol on the one hand, and of those of ammonium nitrate, ammonium chloride, and sodium nitrate as sources of nitrogen, on the other. No nitrite was used in these tests. Moreover, only a limited number of experiments were performed.

The results of these experiments definitely indicated, however, that the production of mutants is feasible in diminished degree with sucrose as well as mannitol. Potassium iodide gave mutants in a solution with ammonium nitrate and with ammonium chloride. Ninhydrin gave variants with both ammonium chloride and sodium nitrate. Chloramine *T* gave changed forms with all three sources of nitrogen. Mutants were obtained with formin and with potassium bichromate. None approached sodium nitrite in effectiveness, however. No mutants were obtained with sodium salicylanilide in the limited number of experiments performed. The type of genetic variant obtained with ninhydrin, chloramine *T,* formin, potassium bichromate and potassium iodide was quite similar to that obtained with sodium nitrite. This also would indicate that the basis of their action is identical with that of nitrite and consists in the elimination of

amino nitrogen from protein. Moreover, no mutants were obtained with many organic compounds chemically without action on amino nitrogen.[4,5] Combination between formaldehyde, liberated from formin, and amino groups would seem to have an action similar to that accompanying destruction of the amino group.

The location of the protein or proteins of the fungus whose assumed change in composition influences its heredity is probably not as easily determined in these forms as in those favorable for cytological study. Whether sodium nitrite, chloramine *T*, ninhydrin, formin, potassium bichromate or potassium iodide can be used to induce genetic variations in green plants, whose cytology can be readily studied, is unknown as yet. It seems possible, however, that a chemical examination of the fungus proteins, either with or without a preliminary separation might lead to further information of value. Amino nitrogen can be estimated by several methods. Also of possible interest in this connection is the correlation found by Van Slyke to exist between the lysine and free amino contents of proteins discussed by Gortner.[2]

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TABLE I. Mutations obtained with species of Aspergilli.

Species of Aspergillus		Normal culture					Extreme mutation culture				
		Fruiting bodies			Aerial Hyphae	Reverse	Fruiting bodies			Aerial hyphae	Reverse
Name	Strain number	Perithecea	Conidia	Sclerotia			Perithecia	Conidia	Sclerotia		
Amstelodami	126	very many	few	none	none	yellow	very few	very many	none	very many	brown
Nidulans Yuill	5616.1	very many	very many	none	few	pink	none.	very few	none	very many	ochre
Fumigatus Yuill	5666a	none	very many	none	none	green	none	none	none	very many	colorless
Alliaceus	200-5506	none	few	very many	none	color-less	none	many	few	very many	yellow
Fischeri	4188.21	very many	few	none	none	color-less	none	very many	none	very many	yellow
Variecolor	5602.3	many	many	none	none	pink	very few	none	none	very many	colorless
Flavus	292-2773	none	few	very many	none	ochre	none	many	none	very many	yellow

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LIBERTY AND MENTAL DEFECT

HOW we are selling our biological heritage for a mess of morons, as charged by anthropologist Hooten, is forcibly demonstrated by clinical psychologist Town* who serves up to view a sorry spectacle of familial feeblemindedness. After extensive psychological examining and investigation of patients at the Lincoln, Ill., School for the Feebleminded and then at the Psychological Clinic of the Buffalo Children's Aid Society where mental deficiency often occurred in sibships, Dr. Town began research as to the intellect of other members of the families of 141 patients in the latter clinic,

*Town, Clara Harrison. *Familial Feeblemindedness.* $2.00. Foster & Stewart, Buffalo, N. Y. 1939.

feebleminded on the basis of psychological tests and clinical diagnosis, whose social and economic histories pointed to multiple familial feeblemindedness. Therefore when possible, clinical examinations were made of others of the consanguinity.

Tables enumerating the familial relationships and sex of these "foci of feeblemindedness" reveal that in the 141 families of 1,384 members, there were at least 398 known feebleminded, — two or more per family. The 180 other members clinically tested were found to be "normal, retarded or borderline,"—all above the moron, though some but slightly so. Since only 42% of the total consanguinity had been available for psychological examinations, of whom over two-thirds were mental defectives, it might well be queried how many more feeble-minded are these families really furnishing the community?

In 61 families there were 150 feebleminded siblings examined; in the other 80 families, there were 69 feebleminded mothers examind and a total of 248 feebleminded members. In 72 families, two generations were represented, in two, three generations. Each member of a family of 12 was feebleminded by clinical measurement and diagnosis.

The dire results to individuals and the serious social import of mental deficiency are vividly pictured by tables descriptive of various defects, mental, physical and social. They show that "These families selected because they are foci of feeblemindedness are also foci of disease, alcoholism, destitution, criminality and vice. The social forces fighting to reduce disease, alcoholism, destitution, criminality and vice, would each and all have greater success did they recognize these families as among the sources of the evils which they are fighting." Dr. Town further deplores the lack of recognition of the great and fundamental disability of the mental defective, — the inability to learn to plan and guide his own life. "Instead of the temporary help, temporary guidance or training which is provided for average individuals who are not adjusting well, every feebleminded person requires supervision and guidance as long as he lives."

This study furnishes no genealogical charts and is of necessity not exhaustive. Patients lacking in intelligence come to crowded mental clinics for help in adjustment, — not for research on heredity, so information is unobtainable or too meagre and incomplete for family trees. All heredity-environment discussion is waived by the author with the practical summation that "Without any assumption concerning why certain families are apparently foci for feeblemindedness, the simple unelaborated fact that they are foci has far-reaching social implications." This is a commonplace to the clinical worker. However, the examples of normal intellects side-by-side with mental defectives, in the same families, in the same sibships, in the same environment, generally bad, — is important evidence against the dictum of some environmentalists that lack of opportunity causes mental defect.

The value of this research to the various disciplines is its emphasis on what every clinical psychologist gleans from first-hand experience in mental measurements, and what every geneticist knows from his laws of heredity,— namely the frequent reoccurrence, though not necessarily universality, of defective intelligence, in the same family. It is hoped that such investigations may lead to more research on the etiology of mental deficiency and to the adoption of preventive measures that are more than slight and ineffectual palliatives. This small volume offers good illustrative as well as scientific material. There are almost insurmountable obstacles in the way of such research but this illuminates glaringly the neglect of science in the solution of practical social problems. Because human beings are involved and because of a failure to interpretate realistically such cliches as freedom and equality, we have the paradox of two incompatible states flourishing simultaneously — civil liberties and mental inferiority.

CLAIRETTE P. ARMSTRONG

A PEDIGREE OF MALFORMED UPPER EXTREMITIES

Showing Variable Dominance

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MANY congenital abnormalities of the upper extremities, both in anatomical deformity and hereditary tendency have been found in the literature by the authors. We have found few cases, however, that were quite so complex as this pedigree.

In the work of Allen B. Kanavel[7] on malformations of the upper extremities, which included sixty cases of hypoplasis, syndactylism, polydactylism, radio-ulnar synostosis, distorted scapula and clavicle, and other lesions, he says, "A hereditary tendency has been noted in some case histories but apparently is not common." He cites H. J. Bagg in his paper and states, "The work of Bagg stands out as one of the great contributions to our basic knowledge of this subject of the theory of the germ plasm origin of these malformations."

Cases of congenital radio-ulnar synostosis have frequently been reported. Cohn[3] in his article entitled "Congenital Bilateral Radio-Ulnar Synostosis" points out that "Radio-Ulnar synostosis is a rare developmental anomaly involving the bones of the forearm. It consists of a bony fusion of the proximal ends of the radius and ulna. It is more common in males and occurs bilaterally in about fifty per cent of the cases. C. B. Davenport, H. L. Nelson, and L. A. Taylor have shown the existence of a definite hereditary tendency."

Milles[12] states that, "Radio-ulnar synostosis and the absence of the long bones have been reported to have an hereditary tendency."

Cohn[4] makes this observation: "Abnormalities of the limbs are definitely inherited and are recessive to the normal." He considers many of the skeletal anomalies due to a retarded development at some particular period.

Probably one of the most important reports in connection with the authors' study is that by Skillern.[17] The patient was five years old and came from healthy, normal parents. He was one of six children all of whom were healthy and well formed. The left upper extremity extends only about one-half the way down the chest wall to which it is adherent. Apparently the only digits present are a rudimentary thumb and a fairly well-developed index finger. The only movements the child can make with the limb are the elevation of the shoulder and some flexion of the rudimentary segments. X-ray pictures revealed a fairly well developed clavicle, very rudimentary acromion, coracoid process, humerus, radius and ulna, carpal and phalangeal bones. This case, especially in the bones of the shoulders and forearm, resembles very much the case being reported.

Philip Lewin[9] reports some interesting cases. One showed a double club hand; absence of both thumbs; four fingers on each hand; radius absent on left arm, and very rudimentary on the right arm; and only three carpal bones present.

Another of Lewin's cases showed in the right arm; no elbow joint, humerus, radius, ulna rudimentary, and only three fingers present. In the left arm there was an ankylosis of the lower arm at right angles to the upper arm. There was no elbow joint and there were only

*The authors wish to express their sincere appreciation to Doctors A. W. Erskine and F. V. Prouty who read the X-ray films.

TWO GENERATIONS OF MALFORMED ARMS

Figure 3

Father and son (II-8 and III-18 in the chart, Figure 9) showing shortened arms and a reduction in the number of fingers. The shoulder girdle is also affected. The arms of the father are about two-thirds normal length and those of the son are less than one-third normal length. The character is definitely inherited though varyingly expressed as an irregular dominant.

three fingers present on each hand.

Although Lewin gives considerable weight to the amnion theory he quotes Boorstein[2] as follows, "Bilateral congenital malformations can generally be traced to heredity while unilateral congenital malformations may be explained on principles of embryonic defects."

Some authors, such as Mercer,[11] and Miller,[13] have reported cases of congenital abnormalities of the extremities and mention heredity as a possible cause for the condition. Ellsworth[6] reports a pedigree of dislocated carpals to be inherited as simple Mendelian dominant. Other workers[15, 18, 14, 5, 19, 10, 8] have reported cases of congenital deformities of the upper appendages and have either failed to give explanations or have concluded that the deformities are due to conditions other than heredity.

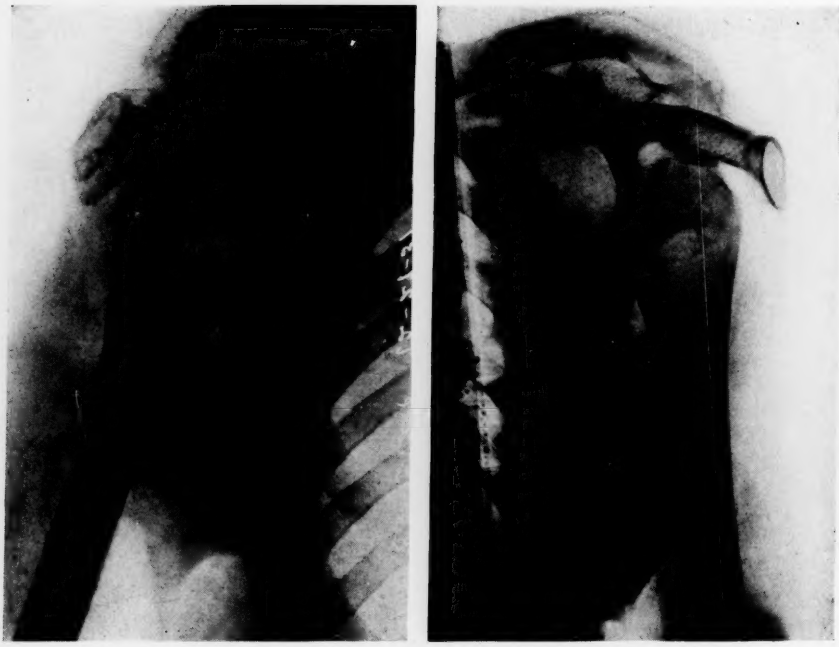
It is not uncommon that hereditary skeletal anomalies exhibit considerable variation, as shown by Stiles.[20] He diverges from the usual explanation of skeletal anomalies, that of either Mendelian dominant or recessive, and reports a case of irregular dominance. In his pedigree he shows that heterozygous individuals may or may not exhibit the malformations.

Other variations from regular dominance of skeletal anomalies have been reported by Wightman,[21] and Baur, Fisher, and Lenz[1] who state that the inheritance of polydactylism is irregularly dominant. Scott[16] also shows a variation from regular dominance of syndactylism.

Inasmuch as the malformation of the upper extremities in this investigation shows so much variation it seems necessary to briefly discuss individually the members of the pedigree, Figure 9.

Beginning with I-2 who exhibits crooked little fingers, his wrists were bent in slightly at birth, showing an ankylosed condition but were comparatively straight in maturity although his wrists were round throughout his life showing a radio-ulnar synostosis.

Through his first wife, I-3, he had three children. One of these, II-6, has the radio-ulnar synostosis condition and a slight mesial bending of the little fingers. Number II-8 from whom many of these data were obtained is shown in Figure 3 with his son, III-18. Both extreme cases of the malformation exhibit short arms, the arms of the father being about two-thirds normal length. His hands are bent inward, caused by an ankylosis of the carpal bones. The right hand has only four fingers as shown in Figure 5 and Figure 6 . The index finger is short and fused with the big finger as far as the first joint of this digit. The third is fused with the fourth finger from the metacarpophalangeal joint half way to the first joint. There is no thumb present. The index finger is slightly "S" shaped. The other three fingers are slightly bent toward the index finger, the fifth finger being bent the most. The joints in the fingers are quite stiff except the metacarpophalangeal joints, which will bend slightly. The X-ray of his hands also shows the radio-ulnar synostosis and Figure 4 shows a peculiar condition in the shoulder girdle. The head of the humerus appears to be "out of joint" but this is due to the inadequate development of the articulating surfaces and to the clavicle which is bowed upward, and the unusual shape of the scapula. This man farms eighty acres of land with little assistance. He is the father of four children. One of these, III-15, is normal and has one normal child, IV-1. III-17 is also normal and has two normal boys, IV-2 and IV-3. Two extremely abnormal cases are III-18 and III-19. One of the boys, III-18, has very short arms as shown in Figure 8, being only about eight inches in length including the hands. There are only three fingers on each hand, corresponding probably to the second, third, and fourth finger. Figure 7 shows how the fourth finger is bent mesially and the other two fingers are bent toward the fourth finger on the left hand. On the right hand the index finger is bent mesially and the other two fingers are bent toward the index finger. On both hands there is some fusion of the skin and flesh between the fingers. Between the fourth and third digits



THE SHOULDERS ARE ALSO DEFORMED

Figure 4

X-ray of humeri and shoulder girdles of II-8. The humeri appear to be dislocated. The clavicles are curved upward and the shape of the scapulae is unusual. Reduced less than the hands of the same individual shown below.

MALFORMED WRISTS AND FINGERS

Figure 5

X-ray of wrists of II-8, showing the deformation of the metacarpal bones in the wrist and the presence of a closely fused first finger on both hands. The finger tips are unusually pointed, and some show claw-like hooks in the bone formation of the fingertips.

DETAIL OF MALFORMED WRISTS OF FATHER

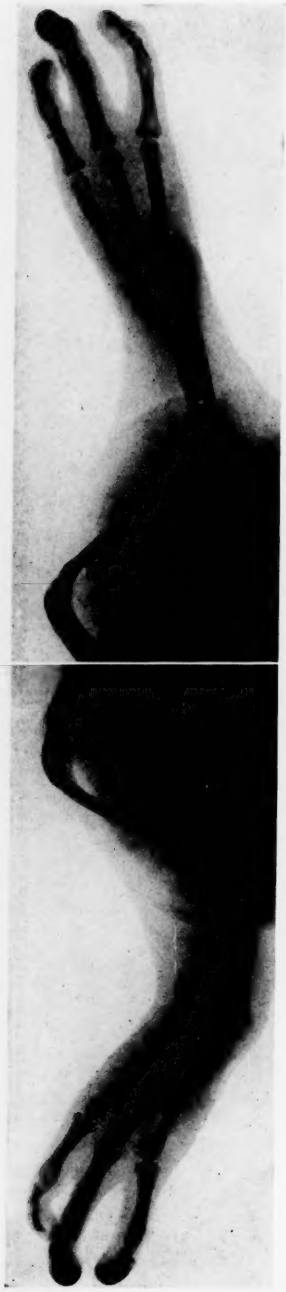
Figure 6

Photograph of abnormal hands of II-8 showing the bending inward caused by an ankylosis of the carpal bones. The right hand has only four fingers, the first two of which are fused together. The left hand has only three fingers evident but the x-ray (Figure 5) shows a fourth closely fused.

there is only a slight fusion at the base of the fingers, but the index and third fingers are fused from the proximal end to nearly the first joint on the index finger. The metacarpophalangeal joints will move slightly but the other joints are stiff. Figure 7 shows the bent clavicles and the deformed scapulae. This boy attends high school, riding a bicycle every day by the aid of ropes which are tied to the handle bars and then wrapped around his short arms.

The brother of the boy discussed above, III-19, had arms which were about the same length as those of III-18. His left hand had three fingers fused, similar to those of III-18. The right hand appeared to have only two fingers, but whether there were really three fingers, two of which were fused, is not known as he died in infancy of pneumonia.

II-6 has ten children. Four of her six sons are normal. One son, III-4, has a webbing between the index finger and thumb on both hands. One son, III-5, has his little fingers bent inward. His hands are slightly bent in and stiffened, and the wrists are round. This boy's condition is nearly the same as his grandfather, I-2. Three of these boys are dead. The four girls in this family are all living and only one of them is normal. The other three, III-10, III-11, and III-12 have their little fingers bent inward. The wrists are quite round but do not



EXTREME MALFORMATION OF SHOULDERS, ARMS, AND HANDS

Figure 7

X-ray of the fin-like arms of III-18 which are the extreme expression in this family of the gene for malformation of arms and shoulders. Only three fingers are present on each hand. The radius and ulna are fused and the humerus is very rudimentary in both arms. The clavicles show marked upward curvature.

bend inward and are not stiff.

I-2, who married the second time to a normal woman had two normal children, II-2 and II-3. Individual II-2 has two children, one of them, III-1, has a webbing between his thumb and index finger on the right hand and the little finger is bent mesially on the left hand. The other child, III-2, has only the left hand deformed. The thumb looks very much like another finger, whether this finger has two or three phalanges could not be learned. Her wrists are slightly bent inward. II-3 has one normal son.

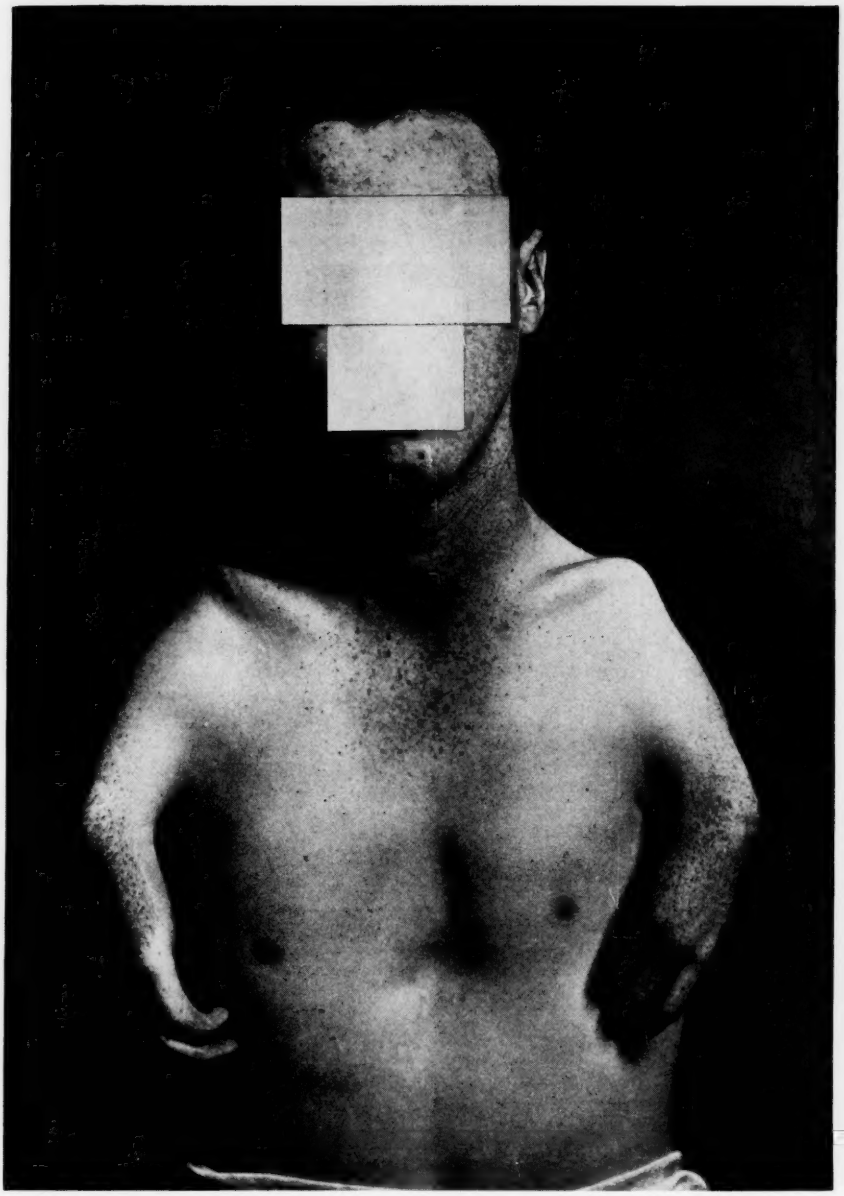
Discussion

The extreme variations of skeletal structures found in this family suggest the complexity of the inheritance of the human skeleton and the important part modifying genes seem to play in its development.

There is good reason to believe that there has been a strong tendency among those interested in human heredity to report only those characters which can be explained easily by known genetical laws. As one goes through the literature on human traits he can scarcely avoid being impressed by the large number which are considered simple recessive or simple dominant. While it is doubtless true that some human characters do appear to be so simply inherited it can well be suspected that if all cases of a given variation had been reported that there would be anything but complete uniformity in the reported mode of inheritance.

The authors believe that our knowledge of human genetics will be most rapidly advanced, not by reporting isolated cases that can be easily analyzed, but rather by reporting all possible types of heredity regardless of whether they lend themselves to a simple explanation or not.

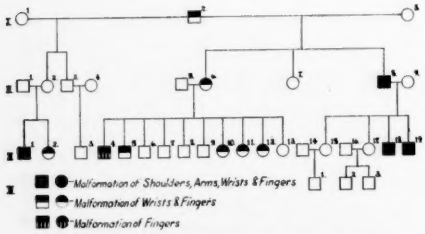
The inheritance of malformed upper extremities here reported is not as easily interpreted as many cases of skeletal anomalies. However, it is suspected that the mode of inheritance in this family, showing such a wide variation in physical expression of a skeletal trait, may be



DETAIL OF FIN-LIKE ARMS OF SON

Figure 8

Photograph of III-18 showing extremely short, fin-like arms with only three fingers on each hand. This is the most extreme expression found in this study of an irregular inheritance of malformation of the upper extremities.



TRANSMISSION OF MALFORMED UPPER EXTREMITIES

Figure 9

Pedigree chart showing the inheritance of malformed hands, wrists, arms and shoulders in a family in which twelve out of twenty-six individuals show the defect. The pedigree shows that the trait is inherited as an irregular dominant.

more typical than has been generally thought.

In this pedigree of malformed upper extremities there are twenty-six individuals in the immediate family. Twelve of these individuals are malformed. This is not a sex-linked character as it appears frequently in both sexes. It cannot be a simple recessive trait as individuals presumably heterozygous show malformations. If individual II-2 were deformed it could be interpreted as a simple dominant character, but since the deformity "skips" generations, irregular dominance is indicated. This means that while the trait is usually a typical dominant it does not always show in the heterozygous condition, presumably as a result of modifying factors.

Summary

1. This study traces skeletal anomalies through three generations.
2. The inheritance of the human skeleton is very complex.
3. Modifying genes often cause hereditary irregularities in the development of the skeleton.
4. It is suspected that the mode of inheritance in this family showing such a wide variation in physical expression of a skeletal trait may be rather typical.
5. An inspection of the pedigree reveals that this trait is not inherited as a simple Mendelian dominant but rather an irregular dominance is indicated.

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EARLY AND VIRESCENT MARIGOLDS*

Two Characters Showing Marked Environmental Effects

THOMAS M. LITTLE, JEROME H. KANTOR,
AND BURR A. ROBINSON, JR.

WHILE *Tagetes erecta*, the common African Marigold, has been cultivated for nearly 400 years and exhibits numerous genetic variations, their inheritance has not been studied very extensively. Punnet† noted briefly the inheritance of doubleness, flower form, and color.

In connection with the development of new varieties of Marigolds at the breeding grounds of the W. Atlee Burpee Company, we have had occasion to study the inheritance of many characters. The present paper deals with two characters which we have had the opportunity of seeing under widely different environmental conditions. The material upon which the studies were made was grown in central and southern California and in Pennsylvania. It was found that the expression of the two genetic factors varied both with the section of the country and the time of the year in which the plants were grown. We were fortunate in having a very large population from which to gather our data. This population, segregating for both characters, consisted of over 5,700 plants.

Material and Methods

The material on which our studies were based came originally from the variety "Yellow Supreme." This variety, when grown in Lompoc, California, in the summer of 1934, bred true for the character we have called "virescent." When the crop of Yellow Supreme was grown in 1935 from seed saved from the previous summer, several normal green plants appeared. These were suspected of being hybrids between Yellow Supreme and some other variety. They were selfed by caging with cheesecloth and the progeny grown the following year in the breeding grounds. The assumption that they were hybrids was substantiated by the fact that they segregated for chlorophyll characters, flower color, flower form, habit, and blooming time. Since the primary purpose of our work was to develop varieties with the form of Yellow Supreme in other colors, plants of apparent commercial value were selected and selfed. These were sent to one of our breeding stations near the Mexican border for testing in the winter of 1936-37. Out of 30 such selections from which we grew large trials, four appeared to be breeding perfectly true. Large blocks were grown from the remnants of these four selections at Lompoc in the summer of 1937. There, instead of breeding true as they had in the winter time, they segregated for foliage characters and blooming time.

The difference in appearance of the populations at Lompoc and in southern California was so pronounced, that we felt an error might have been made in identifying the selections. So further reselections were made and again sent to southern California. All of these seemed quite true for chlorophyll characters and blooming time. The remnants of the two most desirable selections were grown at Lompoc in the summer of 1938. Since these remnants were very large, nearly an acre was grown from each of the two individual plant selections. One of these bred true, but the behavior of the other was the same as that observed the previous summer, i.e., segregation for bloom-

*These investigations were conducted at the Plant Breeding Laboratories of the W. Atlee Burpee Company at Floradale Farms, Lompoc, California, and in southern California and Pennsylvania.

†PUNNETT, R. C. Note on the genetics of the African Marigold (*Tagetes erectus*). *Studia Mendeliana* 187-191. 1923.

EFFECT OF VIRESCENT AND EARLY GENES

Figure 10

Photograph of virescent and normal plants (*A*) shows the stunting of growth when the virescent character is present. The combination of early and virescent genes produces a small plant, compared in *B* to the larger early-normal combination. Virescent and early are both simple Mendelian recessives, independent in inheritance.

ing time and virescent characters.

This segregating progeny contained 5,701 plants which consisted of four phenotypic classes—"Late Normal," "Late Virescent," "Early Normal," and "Early Virescent." The number of each phenotype was counted and recorded.

In addition, 54 progenies were studied which segregated for only the virescent character. These progenies totalled

EARLY MATURING PLANTS ARE SMALLER THAN NORMAL

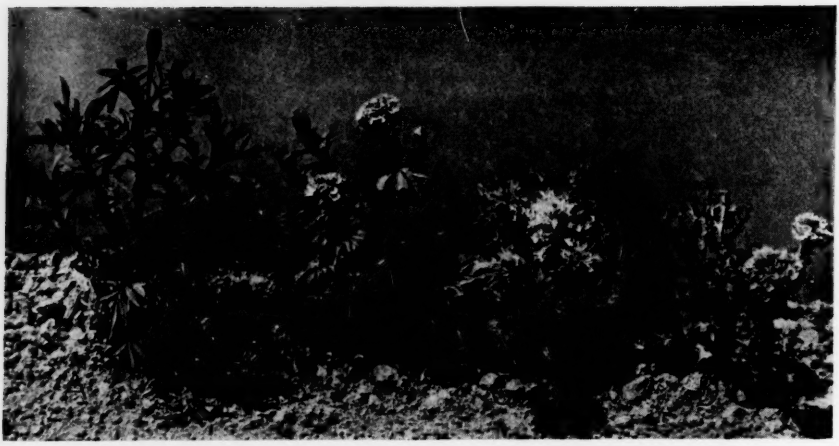
Figure 11

Early and late plants of the same progeny in *A* show the decrease in vigor in the early plant. Long days are necessary for the expression of the early character. *B* compares early and late plants both of which are virescent. Plants homozygous for the recessive early gene blossom about five weeks sooner than the normal plants.

3,957 plants. Moreover, two progenies were studied which segregated for only the early character and these totalled 1,000 plants.

The Virescent Character

Seedlings of virescent plants appear normal, having green cotyledons. However, the true leaves in the young stages



SEGREGATION OF RECESSIVE EARLY AND VIRESCENT GENES

Figure 12

Photograph of all four phenotypes, taken from a progeny segregating for both early and virescent. The plants are: late normal (*A*), early normal (*B*), late virescent (*C*), and early virescent (*D*). Note the relative vigor of the plants. Both the genes reduce survival in homozygous plants, virescent being somewhat more lethal than early.

of plant growth are deficient in chlorophyll, especially around the growing tips. This chlorophyll deficiency also extends to the branches. The leaves later develop chlorophyll, so that as the plants grow larger, the tips of the branches are white and stunted, while the older leaves are normal. When the plant reaches maturity, it assumes a normal appearance with chlorophyll in all the leaves and branches. (Figure 10.)

The virescent character is very pronounced in plants grown in Lompoc, California, in the summer time. Here the temperatures are quite low, ranging from 55 degrees to 75 degrees and seldom exceeding 80 degrees. However, when the plants are grown at Doylestown, Pennsylvania, where the summers are hot, the character is scarcely noticeable. We have also grown plants fifteen miles inland from Lompoc, where the temperatures average 15 to 20 degrees higher, and we could hardly distinguish between virescent and normal plants.

Low temperatures alone, however, will not bring about an expression of the character. Plants grown near the Mexican border in the winter time, where the temperature often drops to near the freezing point and seldom exceeds 70 degrees, never exhibit the virescent character. Evidently length of day plays an important role in affecting expression of the character and the combination of long days and cool weather is essential in differentiating virescent from normal plants.

Early Blooming Plants

The segregating progeny grown at Lompoc in the summer of 1938 was planted in April. In about 12 weeks some of the plants came into bloom while the remainder were not even showing buds. The balance of the population came into bloom in about 17 weeks from the planting time. The early plants were quite dwarf and many were rather weak while the late plants were taller and generally more vigorous (Figure 11). When this same progeny was grown in the winter time, all plants came into bloom at the same time, the length of time required being about 11 weeks.

We have never observed any effect of temperature in bringing about an expression of the blooming time factor, so that long days alone seem to be the only

essential in differentiating the late from early plants.

The early character studied is by no means the only character known in *Tagetes erecta* which affects the blooming time. In our Marigold breeding work at Floradale Farms, we find a complete range of blooming times, from 10 weeks to 22 weeks, showing that there are a great many hereditary factors influencing the blooming time. In the progenies which we studied, one early factor was present without any modifying factors so that the distinction between early and late plants was clear-cut.

Transmission of Virescent and Early Genes

Table I[s]* shows the segregation in the 54 progenies which were segregating for virescence alone. These progenies totalled 3,957 plants of which 78.44% were normal and 21.56% were virescent. The deviation of 3.44% from expectancy is probably due to the fact that the virescent plants are weaker than normal plants and some are probably eliminated in the early seedling stages.

Furthermore, in the large population segregating for both characters (Table III[s]) 78.97% of the plants were normal as to chlorophyll development, and 21.03% were virescent. It is quite evident from these data that virescence is inherited as a simple Mendelian recessive.

Table II[s] shows the results of 500 plants counted in each of two progenies which were segregating for earliness only. In the total of 1,000 plants, 77.3% were late and 22.7% were early. Here, as with the virescent character, there is a deviation from the expectancy which resulted from the death of some of the early plants which are weaker than the late ones. This did not reduce the percentage as much as in the progeny segregating for virescence.

Approximately the same proportion was observed in the large population segregating for both characters, there being 76.87% late plants, while 23.13% of the plants were early. It is evident from the data that earliness is a simple Mendelian character, recessive to lateness.

Virescent and Early Combined

Table III[s] shows the results of the largest progeny studied, which was segregating for both virescence and earliness. Here the results were in close agreement with those found in the progenies segregating for each character alone. 21.03% of the plants were virescent and 23.13% were early. Furthermore, the data show that the two characters are inherited independently. Taking into consideration the partial lethal effect of the characters as shown by their segregation in progenies where they were found alone, the observed data for a progeny in which they are both segregating (60.87% Late Normal: 16.00% Late Virescent: 18.10% Early Normal: 5.03% Early Virescent) are in close agreement with the expectancy in a 9:3:3:1 ratio corrected for the lethal effect (60.63% Late Normal: 16.67% Late Virescent: 17.81% Early Normal: 4.89% Early Virescent) as shown in Table IV[s].

Summary

1. Virescent, in *Tagetes erecta,* is a character in which the development of chlorophyll in the stems and leaves is delayed.

2. Long days and cool weather are necessary to bring about an expression of the virescent character.

3. Virescent is inherited as a simple Mendelian recessive.

4. The virescent character acts as a partial lethal, and some of the plants die as a result of their chlorophyll deficiency before they reach maturity.

5. The early character studied causes

*The four detailed tables have been afforded supplementary publication as Document No. 1326 of the American Documentation Institute, 2101 Constitution Ave., N. W., Washington, D. C. Enclose 22 cents for the tables on microfilm, enlarged to readable size with hand-viewer, or 40 cents for photoprints, readable without mechanical aid.

the plants to come into bloom in about 12 weeks, whereas normal plants come into bloom in about 17 weeks, when grown in the summer time.

6. Long days are necessary to bring about an expression of the early character, and during short days early and late plants bloom at the same time.

7. Earliness is inherited as a simple Mendelian recessive.

8. The early plants are somewhat weaker than late ones so that the early character acts as a partial lethal.

9. Virescent and early are inherited independently, showing no linkage when segregating in the same progeny.

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WHITE-FLOWER CHARACTER FROM X-RAY TREATMENT OF TOMATO SEED*

P. A. Young

In attempting to develop tomato varieties that would be resistant to certain diseases, a new white-flowered variation, presumably a mutation, was discovered at the Tomato Disease Laboratory of the Texas Agricultural Experiment Station at Jacksonville in 1938. Instead of having ordinary yellow corollas, the new selections have corollas that are pure white or only slightly streaked with yellow. These white-flowered selections resemble Marglobe tomatoes in all other characters. They originated from Marglobe tomato seeds that were treated with X-rays in 1933 and grown in 1934 by the late Dr. J. J. Taubenhaus.[1] Seed from this 1934 crop (T5) produced only yellow-flowered plants when tested by the writer from 1936 to 1939.

Table I[s]† gives data on the behavior of single-plant selections of three generations of progeny of this T5 Marglobe tomato seed. Selection T161 segregated with very nearly three-fourths of its plants having yellow flowers and one-fourth of its plants having white flowers, and T335 segregated almost identically, thus showing they were heterozygous for flower color.[2] In contrast, selection T339 produced 85 plants with yellow flowers and only one plant with white flowers. The one white-flowered plant probably resulted from a mutation. This explanation apparently applies also to the one white-flowered plant of T162.

Seed was selected separately from 10 of the white-flowered plants of T161. Six of these selections produced 510 plants all of which bore white flowers. Three other selections (T336, T337, and T340) produced 445 plants of which 98.2 per cent had white flowers and only 1.8 per cent had yellow flowers. Their white-flowered parents grew within a few feet of many yellow-flowered tomatoes, so the few yellow-flowered plants probably resulted from natural cross-pollination. Similarly, seed of four fruits of selection T378 (T444 to T447) produced 287 plants, 99.3 per cent of which bore white flowers. Natural cross-pollination probably explains the 0.7 per cent of plants with yellow flowers, as pollen of yellow flowers sometimes was liberated in the air when flowers were artificially crossed in the greenhouse where the plants of T378 grew.

Selection T338 behaved unlike the others in that it segregated in a 1:1 ratio of yellow-flowered and white-flowered plants. This probably resulted from a natural backcross of a white-flowered plant with a heterozygous yellow-flowered plant of T161 in the field.

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*Technical Paper No. 549 of the Texas Agricultural Experiment Station, College Station, Texas.

†Table I has been afforded supplementary publication as A. D. I. Document No. 1327. Enclose $0.21 for the table on microfilm or $0.30 for a photoprint. (See page 77 for details.)

A white-flowered selection (T378) was crossed with ordinary yellow-flowered Rutgers, Marglobe, and Louisiana Red tomatoes in the greenhouse. All of the crossed flowers were bagged. Their 151 progeny plants (T436 to T441) all bore yellow flowers in the F_1 generation.

A summary of the evidence in Table I[s] shows that the yellow-flower character (Y) is apparently a simple dominant over the recessive white-flower character (y). These characters segregated in practically a 3:1 Mendelian ratio in the 159 plants of T161 and T335. The white-flowered plants thus appear to be homozygous recessive for flower color. Except for a few plants, 13 selections bred true for the white-flower character in the two generations tested. The crosses with yellow-flowered varieties produced all yellow-flowered plants in the F_1 generation and thus added to the evidence of the dominance of the yellow-flower character.

The white-flower character promises to be a valuable marker for selections prominently resistant to certain diseases, and this idea has been included in a program of breeding for disease resistance. This character should be particularly valuable as such a marker since white-flowered selections already compare favorably in yields and in resistance to wilt (caused by *Fusarium lycopersici*) with Marglobe, Rutgers, Riverside, Louisiana Red, and other wilt-resistant varieties. Like Marglobe, white-flowered selections are resistant to blossom-end rot.

Summary

White-flowered tomatoes were first observed in 1938. The white-flower character presumably originated by mutation stimulated by X-ray treatment of the ancestor Marglobe tomato seed. The yellow-flower character (Y) is dominant over the recessive white-flower character (y). These characters segregated in a 3:1 Mendelian ratio. White-flowered plants apparently are homozygous recessive for white flowers, and bred true through two generations tested. The white-flower character promises to be a valuable marker for disease resistant selections.

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MIGRATION AND PHYSIQUE

VERY few studies have ever been made on the effect of a changed environment upon the physical characteristics of a migrating people. The pioneer investigation of Boas on the changes in bodily form of immigrants to the United States has been almost alone in this field. The conclusions of Boas have been widely quoted and have encountered quite a little adverse criticism on the score of methodology. Further invesigation of the problem, therefore, has long been a desideratum.

It is well known that stature, pigmentation, and many other physical traits are affected by environmental forces, but little is known of the extent of the changes thus produced. How far the physical traits which anthropologists commonly employ in distinguishing races persist under a changed environment is for the most part unknown. It is therefore desirable that these criteria of racial distinctiveness be subjected to a critical test. This task Dr. Shapiro has assayed to carry out on the basis of elaborate measurements of Japanese migrants to Hawaii.* The material was well chosen for such an undertaking, inasmuch as measurements were made not only of the immigrants and their children, but of the relatives of these immigrants still liv-

*H. L. Shapiro: Migration and Environment (Oxford, University Press), pp. xi + 594. 1939.

ing in Japan. Since the latter live in the regions from which the immigrants came and belong to the same socio-economic groups, they constitute the most satisfactory basis for comparison that could well be selected. The number of individuals measured was large enough to give many statistically reliable results, since it consisted of 2,594 subjects, of which 1,713 were adults, fairly evenly divided into immigrants, children of immigrants, and relatives in Japan.

"The immigrant males," according to Shapiro, "are on the average heavier than the sedentes; they have shorter trunks and longer legs; their distal arm segments are longer; their shoulders are wider; their chests are broader but shallower. In the structure of the face and head the immigrant men again reveal profound differences from their parental type. They have higher heads, wider bigonial diameters, longer total and upper face heights, greater dimensions for nose height and length, less salient nasal tips, narrower noses, narrower inter-ocular and wider bi-ocular diameters. These are all statistically significant differentiations." In general, immigrant females show similar differences, but with some notable exceptions.

Since the immigrants were mainly adults at the time of their arrival in Hawaii, Shapiro attributes their physical differences, with the exception of weight, to selection. The migrating individuals were much the same in type, regardless of the regions of Japan from which they came. The causes which determine the selection of the immigrant type are not clear. Various conjectures were discussed, but no satisfactory solution of the problem was arrived at.

The Japanese born in Hawaii showed significant differences from the original migrants. The males averaged 4.11 cm. higher in stature, whereas the stature of the immigrants and their relatives in Japan was practically the same. The increase in stature of females was less, namely, 1.71 cm. While the changes in the two sexes were in the same direction in several physical traits and proportions, there were several others which were divergent. In general, the Hawaiian-born deviated from the Japanese residents in the same direction as the original migrants, and to a still greater extent, but there were exceptions also to this rule.

Shapiro's work is by far the most detailed study that has been made of the biological effects of migration. Of its 594 pages 392 consist of tables of measurements, and there are over 50 pages of graphs representing, chiefly, changes due to age. As a sort of compensatory lack, there is no index.—S. J. HOLMES.

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A NEW TYPE OF BOVINE AGNATHIA

RECENTLY, an unusual type of lethal agnathia was brought to the Veterinary Hospital of Oka (LaTrappe, Prov. of Quebec, Canada) by a local farmer.

Last March, Ely, Hull and Morrison[3] gave a detailed account of cases of a lethal "jawlessness" which is characterized by them as being entirely new and hereditary. They mention four cases, three of which are of the same parentage. The four Jersey calves which were born dead are described as having the following type of lethal agnathia:

1. The lower jaw of the monster is 8 cm. shorter than the upper one.

2. Under the ears hang two wattle-like appendages (5 cm. long) bearing large coarse black hairs.

3. The mandible consists of soft osseous tooth tissues, crowded together into an irregular mass that fills the mandibular space and separates the epiglottis from the posterior part of the soft palate.

4. The lower lip does not extend past the first upper premolar.

5. An X-ray picture does not reveal any evidence of incisor teeth.

6. The tongue is pushed anteriorly and is quite small.

7. The coronoid process of the mandible is absent; no trace of hyoid bone.

8. There is a complete ankylosis of the temporo-mandibular articulation.

HEAD OF AGNATHIC CALF
Figure 13

Photographs of the bottom view (*A*) and the sagittal section (*B*) of the head of the calf. In *A* the rudimentary lower jaw has been dissected to uncover the teeth. The sagittal section shows: *a*—rudimentary tongue, inserted originally under the palate, through the nasal cavity; *b*—lower jaw with a well-developed incisor (*c*) uncovered.

The case of the Ayrshire calf brought to the Veterinary Hospital of Oka (Figure 13) is briefly as follows:

1. The upper jaw is 10 cm. longer than the lower one.
2. No trace of wattle appendages.
3. At the bottom of the wide open mouth, two rudimentary mandibles may be seen having no connection at their extremity. They are composed of soft tissues covered by a mucous. The molars and incisors are well formed and seemingly at the verge of appearance.
4. The temporo-mandibular articulation is very rudimentary and somewhat in the state of ankylosis.
5. The coronoid apophysis is absent.
6. The tongue is very narrow and small, hardly 5 cm. in length and, instead of lying at the bottom of the mouth, as in the preceding case, it is uplifted and inserted under the palate through the nasal cavity.
7. Having only the head, it was impossible for us to determine the sex of the specimen.

This case, up to date, being met only once, it is actually not only impossible to say whether the factor here in action is recessive, should the abnormality be hereditary.

The Oka Ayrshire case is very different from the cases of Ely, Hull and Morrison and from those cited by Mohr,[6] Williams,[7] and Hutt.[4] It is also different from the recessive lethal "parrot bill" described by C. Wriedt,[8] in which the lower jaw is entirely wanting, and the bone of the upper one strongly bent over the opened mouth.

Very recently, H. E. Annett[1] published a new type of agnathia found in five descendants, three heifers and two bull calves, of a Shorthorn bull of the milking breed, in New Zealand. The abnormality consisted in a deformity of the lower jaw, which was only half the normal length. The teeth were normal and the tongue, having no support, hung straight down outside the mouth. The calves appeared to be normal in every respect." The gene must be recessive and the heterozygote is indistinguishable from normal. As everybody may note, this last case is also different from ours. The occurrence of three heifers suffering of this type of agnathia is a new feature the agnathia lethal having been observed heretofore only in the male sex.

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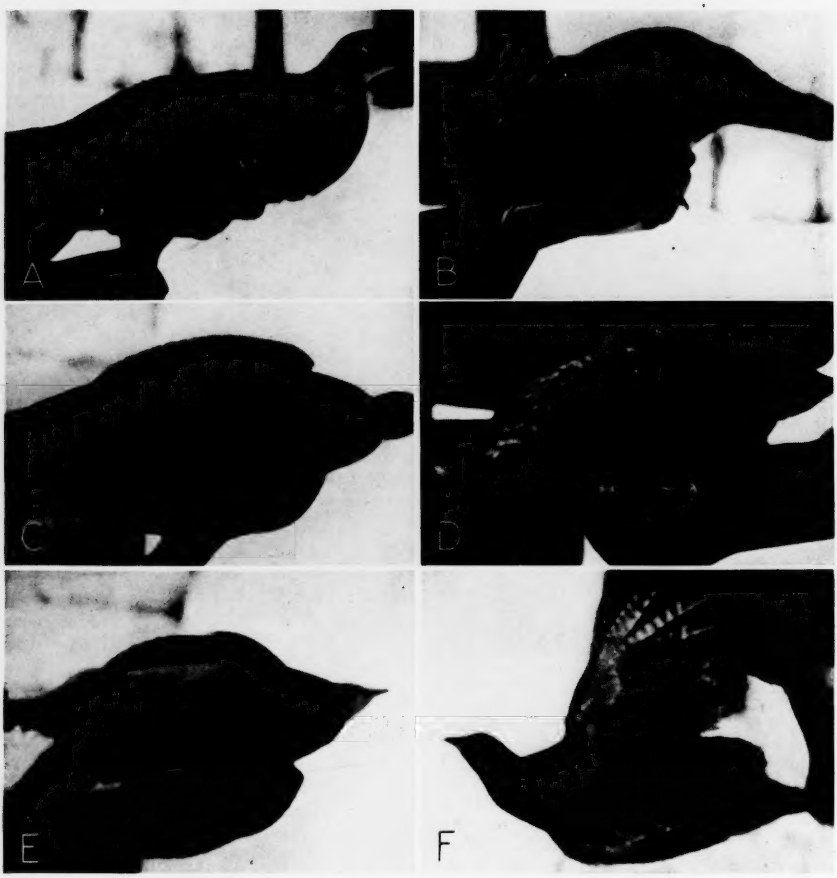

NORMAL AND "MULE" PHEASANTS COMPARED

Figure 14

Photographs of three views of a normal ring-neck pheasant (*A, C, E*) compared with three views of "Mule" Pheasant II (*B, D, F*). "Mule" pheasants, with mixed male and female plumage characters are found occasionally among hen pheasants on game farms. Note the incomplete black ring on the neck of the "mule" and the dark, male-like feathers scattered over the back and breast.

THE "MULE" PHEASANT

Sex Invert or Intersex*

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OCCASIONALLY pheasants occur whose size is that of the hen but whose plumage is mixed male and female. In ring-necked pheasants (*Phasianus colchicus torquatus*), such individuals are partially ring-necked and have tail feathers somewhat like short male feathers. The neck, breast and belly feathers are mostly male in character with many female feathers interspersed. To a less degree this is true also of the shoulder regions. On other parts of the body female types of feathers predominate. Such birds are readily distinguished from normal males and females and are called "mule" pheasants by the breeders and men of the State Departments of Fish and Game.

The nick-name "mule" is given probably because after assuming their unusual plumage they fail to lay any more eggs and are useless as breeders. In some cases they are reputed to have possessed normal plumage and to have laid eggs as apparently normal hens before acquiring the mixed plumage. As will be seen from what follows, the histological findings with regard to the sex-glands of one case (October) suggest such a possibility and in the second case (January) do not entirely exclude such an interpretation.

The condition was first brought to our attention by Mr. Bronson of the State Department of Fish and Game, who sent us the skinned body of one such bird two days after it was found dead on October 3, 1938. With the body came a report that it was believed that the bird had laid eggs during the previous summer before assuming the intersexual plumage. It had then appeared to be a normal female, in both plumage and behavior. This could not be checked further as records failed to show evidence on this point. Its plumage was not available for study.

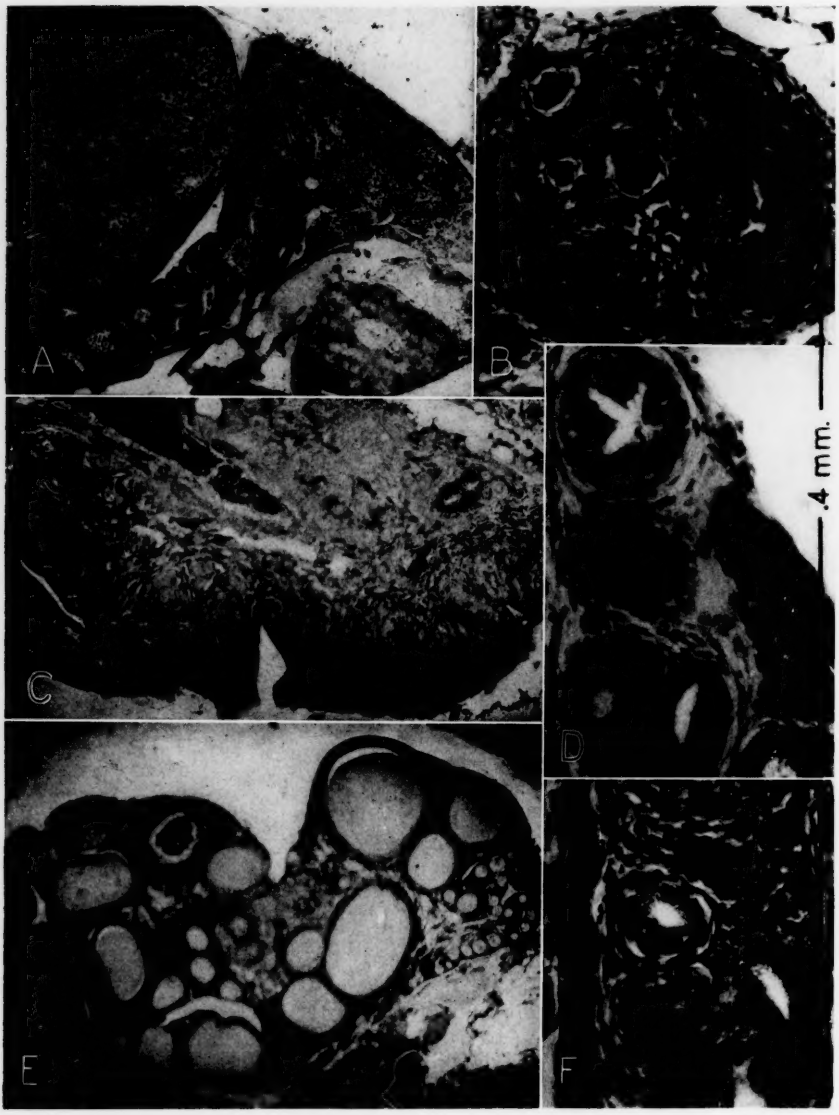
In January, 1939, another "mule" was found among some pheasants held for "planting" at Shade Swamp Sanctuary, Farmington, Connecticut. Dr. R. P. Hunter, State Superintendent of Fish and Game, kindly permitted us to secure this bird and a normal hen pheasant of similar age and variety, for histological study and comparison of the sex-glands associated with this condition and that of the normal female.

Material and Methods

The birds mentioned above were used in this study. One was secured two days after death on October 3. Its kidneys with adrenals and ovaries attached were removed whole and fixed in Bouin's fluid as soon as possible. But the tissues had already undergone necrotic changes before fixation. Only gross structure could be made out. Photographs of this bird were not obtained. The other "mule" and the normal hen were secured alive and apparently healthy on January 20, 1939, photographed from three aspects, and killed by cutting through the roof of the mouth into the brain. They were skinned and the complete plumage preserved for future study. The sexual organs and adrenals attached to the kidneys were removed and fixed in Bouin's fluid at once.

After trimming away most of the kidneys, the rest with the sex-organs and adrenals of all three birds were serially sectioned transversely from paraffin at 10μ, stained in iron-hematoxylin and faint eosin and mounted serially in dam-

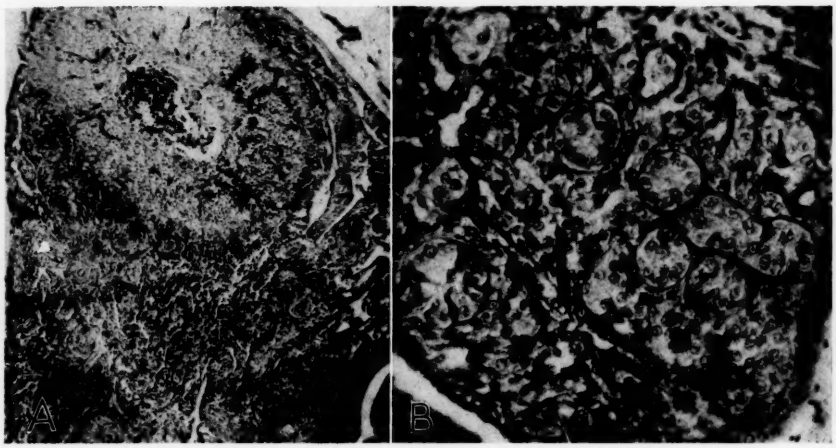
*Aided by a grant from the Penrose Fund of the American Philosophical Society, 1938-9.



SECTIONS OF OVARIES OF TWO "MULE" PHEASANTS AND OF A NORMAL FEMALE

Figure 15

Photomicrographs are shown of sections through two parts of the ovary of the two "mule" pheasants discussed in this article (*A*, *C*), and of a normal pheasant for comparison (*E*). The ovary of both mules showed marked contrast to the normal female. "Mule" No. 1 was not examined until two days after death, but the histological situation even then is quite clear. *A* shows a section comparable to the normal control (*E*). The clear areas perhaps represent the site of old follicles. No active follicle production was evident at the time of death. *B* shows the region of the ovary which had undergone transformation in the male direction. The tissue was not in good condition but the resemblance to *D* is obvious. This structure closely parallels normal testicular development, being strongly suggestive of a section through the male epididy-



MALE AND FEMALE TRAITS IN "MULE" OVARY

Figure 16

Photomicrographs of two sections of the ovary of "Mule" No. II. *A* shows two regions resembling old corpora lutea or atretic follicles, the only suggestions that this tissue has been derived from a normal ovary containing eggs and follicles. The section of a nest of tubules in *B* shows male-like Sertoli cells in a stage of early activation. *A* × 84, *B* × 335.

mar, after the usual paraffin technique. After study of the slides, typical sections were photomicrographed at various magnifications as noted in the legends for plates from the most significant, for comparison.

Observations*

Plumage

Comparison of the normal hen (Figure 14 *A, C, E*) with the "mule" (Figure 14 *B, D, F*), both of January 20, show that the "mule" feathering is intermediate between those of the normal male and female. Feathers of both sex-types are intermingled with intergrades from almost male character to almost female. The "mule" does not appear to be a gynandromorph; but rather an intersex or sex-invert in which feathering in different regions has given different responses to a peculiar internal ecology of sex-hormone imbalance. The greatest degrees of maleness of feathering appear on neck, breast, belly, shoulders and tail. More complete description of feather types and distribution is reserved for a later time when more specimens are available.

Sex-glands

Gross dissection showed nothing resembling a testis in either of the "mules." The sex-glands appeared to be small left ovaries without visible follicles or "eggs," occupying the usual place. No unusual growth of the rudimentary right gland was evident. Neither it nor the oviduct could be seen and the latter was not secured for study. In the normal hen it was readily found and secured, but showed nothing unusual and resembled those of starlings or sparrows of like time of year except

LEGEND TO FIGURE 15—Continued

mis. *C* shows the ovary of Mule II which gives clear evidence that the same situation existed. Here the body of the ovary is dormant and is accompanied by some development of the epididymal region (*D*). These sections are to be compared with *E* and *F* which show the same regions for the normal ovary. Here there is latent male tissue present but this is definitely in a resting stage. Notice that the ducts are much smaller and most of them much less clearly differentiated. The functional ovarian tissue (*E*) on the other hand is filled with eggs and large follicles in process of development. The relative enlargement of each section is shown by the arrows, each of which represents 0.4 mm. (*A*, 84×; *C* and *E* 33.5×, and *B, D* and *F*, 335×.) Thus the epididymal ducts are in reality very much smaller than the follicles in the normal ovary.

*Additional photomicrographs of sections of the ovaries of the three pheasants described herewith have been published as A. D. I. Document No. 1325. Remit 23 cents for copies on microfilm and 50 cents for photoprints. (See page 77 for details.)

for larger size. The cross-section shape of the "mule" ovary of January 20 is shown in Figure 15*C*. It is quite evidently that of an ovary. That of the October "mule" was similar but considerably larger, with grooves and ridges or hillocks which are not entirely lacking from the January one. (See Figure 15*A*.) These grooves and hillocks are also found on the normal ovary (Figure 15*E*); but not in a normal testis. So the gross conformation of the sex-glands of both "mules" is that of an ovary and female in type.

Histologically neither "mule" gonad exhibited any "eggs" or characteristic follicles. In the October one (Figure 15*A*), there were clear, apparently fluid-filled, regions in section which may have been either cysts or atretic or degenerating follicles. Because of the necrotic condition of the whole gland it is impossible to be sure which they were. This doubt makes it appear possible that the story of this bird's having laid eggs in the previous summer might be true. Pigmentation is not very marked in this gonad. The black flecks seen in the figures are floating chips of hard dense blood-clots from adjacent vessels fragmented in cutting. One part of the series of sections suggests the rounding up of a testis-like ridge with tunica albuginea round it; but the condition of the tissues makes it impossible to know whether or not its interior was made up of sex-cords or tubules. The cyst-like elements in this part of the gonad are small, the larger ones being confined to other parts of the gonad. Various sizes of these cysts suggest follicles caught in degeneration at different stages. But they may be pockets of pus or fluids or bacterial aggregates.

The January 20th "mule" ovary (Figure 15*C* and 16*A*), fixed immediately after killing, shows only two regions that suggest the possible presence of ovarian follicles preceding the assumption of the "mule" condition (Figure 16*A*). These resemble old corpora lutea undergoing regression. One exhibits a loose core either heavily pigmented or with a blood clot undergoing resorption. In the other, pigmentation is marked around the margins of a central, more homogeneous, core. There is no evidence of a tendency to organize a testis-like ridge; but ridges and folds extend lengthwise on the gland. There are also some remnants of what may have been a capsule round parts of the gland simulating an infundibulum or oviductal funnel. This is also seen round the normal ovary (Figure 15*E*). The whole gland gives evidence of the ingrowth of cords from the outside or peritoneal surface and their tendency to coil in the cortex-like region, causing the heavy pigment masses of this gland to be stretched out and come to lie in the interstitial regions between these cords. In some regions these cords seem to have pushed the pigment into the medulla of the gland leaving regions almost devoid of it. However, it is also possible that the pigment may have penetrated from the medulla into the cortex-like region and between the tubules, or it may have been laid down *in situ* after the cords came. This pigment is also present in certain regions of the normal ovary around some follicles (Figure 15*E*).

The most significant condition of the ovary of the January "mule" is the presence in many regions of seminiferous or testicular tubules derived from the above-mentioned cords. These are well shown in Figure 15*C* at low magnification, and particularly well in Figure 16*B* at 335 diameters. Figure 16*B* shows a good nest of such tubules with Sertoli-like cells well shown in one tubule in the lower left corner of the figure and isolated from the nest of such tubules.

The ovary of the normal hen pheasant on January 20 is full of "eggs" and follicles in various stages of development and maturation (Figure 15*E*). Figure 15*E* is at the same magnification as Figure 15*C* of the "mule" pheasant. Comparison shows clearly the amount of masculinization of the "mule" ovary and its complete loss of all femaleness except its shape and position. Its pigmentation is also evident.

Epididymides

Both "mules" and the normal hen pheasant have what appears to be the anatomical equivalent of an epididymis and vas deferens. The relations of this structure to the sex-gland and its histological structure are shown in Figure 15*B*, for the October "mule," Figure 15*D* for the January one and Figure 15*F* for the hen pheasant. These are at the same magnification (335) for the two "mules" and the hen, respectively. They indicate that this structure is present in the adult female and needs not to be created anew in the "mule." It is perhaps more highly developed in the "mule."

Discussion

Whether these "mules" are cases of sex-reversal, sex-inversion, or of intersexuality in the sense of Goldschmidt it is impossible to state with certainty. They resemble in some respects the freemartin among cattle, pigs and goats, or the newer experimental freemartins or intersexes among chickens induced by adding sex hormones to the incubating egg and in mammals by continued influence of such hormones injected before or after birth. In any case they represent a form of aberrant sexual heredity and development or retrogression.

Crew's[2] cases in the fowl, also discussed by Fell,[6] are similar to these in some respects. Krediet[14] has recently described and discussed a case of sex-reversal from female to male in a cow at the age of three years. Cases of early

removal of the left ovary in the fowl result in changes of plumage, more or less permanent, associated with reactivation of the right gonad-rudiment, and sometimes of remnants on the left side, into a testis-like gland (Benoit,[1] Zavadowsky,[21] Domm,[5] and Lillie[15]). But in these pheasants it is the left gonad *in situ* that undergoes masculinization and presumably induces or permits the change toward maleness of plumage (the fundamental type for such birds). Any change in the rudimentary right gonad that may have occurred was so slight as not to render that structure identifiable in these cases, whether "mules" or normal hen. It is perhaps safe to conclude that none occurred, and that such change was confined to the left gland.

In some of the reactivated right gonads of fowl late stages of spermatogenesis were noted. In these "mules" none was shown. Nevertheless, the apparent early stages of activation of the Sertoli-like cells (Figure 16*B* suggest that spermatogenesis may occur in the normal breeding season in these "mules"). Cases to test this, and the effects of increased light at the breeding season are being sought. Winter lighting will also be tried on other fall and winter cases. This will settle more completely the question of the completeness of reversal or masculinization of such birds. All the necessary cellular elements for spermatogenesis seem to be present in the "ovary" of the January "mule" awaiting activation.

Fell's[6] cases indicate that rarely the left ovary of the fowl, like these "mules" has the capacity to produce sex-cords of the male type. She attributed these cases to lack of balance of hereditary factors. Lillie[15] discussed the bearing of these cases and that of Riddle's[18] sex-reversed pigeon upon the problem of sex-inversion in the hen. We can add little to such discussion from these two cases, only one of which is well enough preserved to make its fundamental structure discernable. This is also particularly difficult in a hybrid race like the ring-necked pheasant in which the genetic basis for sexuality is little known except for the evidence that it is very variable. These are but two more cases for the accumulation of data needed for a more complete elucidation of the problem.

The presence of the rudiment of an epididymis and vas deferens on the left side of the normal hen pheasant as well as in the "mules" point to the possibility of functional activity as males, if proper rete and vas efferential elements develop. These gonads give no clear signs of these structures. They might show up in the breeding season or under night-lighting, when the whole reproductive complex is at the climax of activity and development.

The intersexual or sex-inverted condition of these pheasants can hardly be hormone-induced beyond the plumage and accesories, as were the intersexes of Dantchakoff,[4] Willier,[19] among birds, or Dantchakoff,[3] Greene and Ivy,[7] Greene, Burrell and Ivy,[8,9] Raynaud,[16,17] and others, among mammals. No mechanism for such induction can be even postulated as operating here.

Summary

1. Two cases of "mule," intersexual or sex-inverted pheasants are described and compared with a normal hen pheasant.

2. "Mule" plumage is intermediate between those of normal males and females.

3. The left "ovary" of the "mule" is without "eggs" or ovarian follicles, either lost or never developed, and the January one possesses sex-cords and tubules of the male type in which Sertoli-like cells give signs of early activation.

4. In both "mule" ovaries, certain regions may permit interpretation as resulting from regression of previously existing ovarian follicles or as cysts or pus pockets.

5. Right gonad rudiments were not identified, so no evidence of their activation was noted.

6. Both "mules" and normal hen pheasants possess homologues of left epididymis and vas deferens.

7. Some features of these birds in relation to the problem of intersexuality or sex-inversion in birds and mammals are pointed out.

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The Chantecler Breed of Poultry

THE practical application of genetics has been well demonstrated by the synthesis of the "all-Canadian" breed of poultry, the Chantecler, recorded by Professor Leon J. Cole in this JOURNAL in April, 1920. The story began in 1908 when Brother M. Wilfrid, a Trappist monk and professor at the Oka Agricultural Institute of La Trappe, Quebec, Canada, set out to produce a bird that would be physically adaptable to the rigorous Canadian winters. Combining the genes of the Cornish, Leghorn, Rhode Island Red, Wyandotte, and White Plymouth Rock, he produced two birds with heavy plumage and small combs and wattles. These two birds and their F_1 offspring were recognized as a breed by the American Poultry Association in 1920. Although the breed showed some variation at first, the birds now chosen as the best examples of the breed closely resemble the two original birds pictured in this JOURNAL in 1922.

The originator of the new breed has recently written a guide* to poultry breeding combined with a monograph of the Chantecler. In the first six sections he discusses care, feeding and housing of poultry; including notes on the selection of proper cocks and breeding hens. The seventh section is mainly devoted to the history of the Chantecler, and to a detailed list of its distinctive characters.—C. B.

*Wilfrid, Frere M., Le Guide Avicole, L'Institut Agricole D'Oka, P. Q. Canada. 132 pp. 1938.

A GENETIC HISTORY OF HAMPSHIRE SHEEP*

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THE systems of breeding used in producing the various breeds of farm animals are especially interesting, because so many analogies have been drawn between what happens when man is consciously struggling to perfect his four-footed servants and what happens in evolution in nature. The breeding systems used by the average breeder have already been studied in several breeds of cattle, two breeds of swine, one breed of horses and one breed of sheep and these studies have yielded a general picture which is surprisingly similar for all except the very early history of the Shorthorns. The Hampshire sheep was chosen for a second study of this kind on sheep because it is one of the most important breeds in this country and because of personal curiosity about its history.

The Beginnings of the Breed

The Hampshire breed originated in the counties of Wilts, Hants, and Berks in southern England. This region (see Figure 17) is a series of low lying, fairly fertile chalk hills popularly known as the West County Downs. Two native breeds or races, the Wiltshire Horned sheep and the Berkshire Knots, occupied this area prior to 1830.[7] These breeds had little to commend them except hardiness to adverse environmental conditions. After the enclosure of the commons, feeding practices improved greatly. This, combined with the demand for better mutton, caused breeders to attempt to improve their sheep. Most of them crossed their flocks with the improved Southdown which had developed in Sussex just to the east. The most prominent of the early breed improvers was Wm. Humphries. By consistent line breeding to two rams, within a flock closed to outside blood, he was able to fix the type of his flock fairly uniformly. Figures 18 and 19 show artists' conceptions of some of the ancestors of the modern Hampshires.

The Hampshire Down Sheep Breeders Association, in England, was organized in 1889 and the first volume of the flock book was issued in 1890. Flocks were entered as a unit after inspection. The rams were registered individually, but unless the ewes were to be exported they were identified only by the flocks in which they were born. Since 1908, only animals whose sire and dam were from registered flocks have been eligible for registry, thus closing the herdbook against outside blood.

Hampshires in the United States

Several flocks of Hampshire sheep (perhaps the old Wiltshire breed) were established in Virginia prior to 1860 but none of these survived the war[2] as flocks with known pedigrees. A small flock was imported to Long Island in 1855 and a few other importations were made from 1865 to 1890. The American Hampshire Down Sheep Breeders Association was organized in 1889, the same year in which the English Association was organized. Volume one of the American flock book was issued in 1890. It listed only 35 members with 739 sheep. Both rams and ewes were given individual registration. Only animals which traced in all lines to recognized English flocks could be entered. No provision was ever made for registration of grades in the United States.

As concerns numbers the Hampshire breed grew rather slowly in the United States until after 1906. From 1906 to 1912 a great increase in numbers took place, principally by importations from England. The peak of importation was reached in 1909 when one firm alone

*Journal Paper No. J-640 of the Iowa Agricultural Experiment Station, Ames, Iowa.

SHEEP COUNTRY OF OLD ENGLAND

Figure 17

Showing the region in which the Hampshire and neighboring breeds developed.

imported over 1,000 head. Although importations fell off after 1912, numbers continued to increase until 1927 during which year about 26,000 head were registered. After 1927 the breed seems to have come near an equilibrium in number, with about 20,000 head being registered annually.

Methods of Study

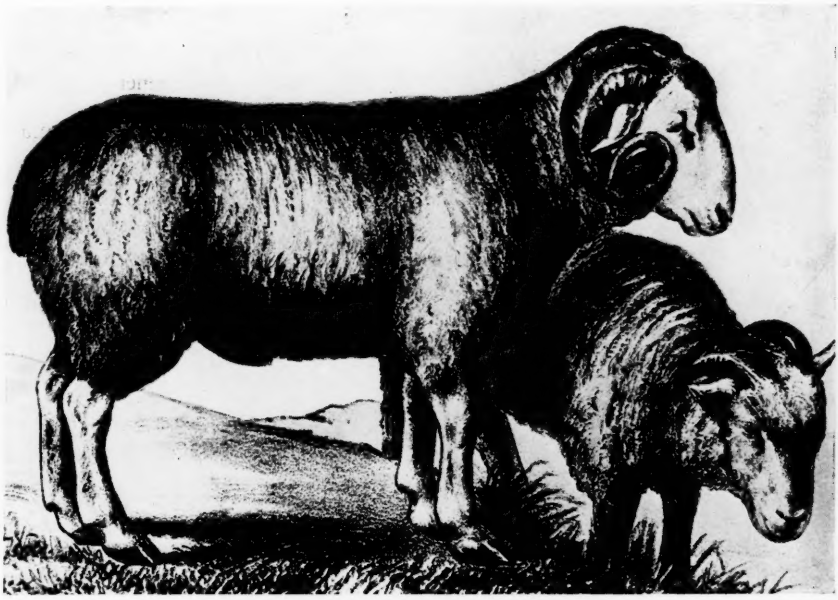
Willham[8] has recently reviewed the published studies made by the methods used here. Besides the studies he mentioned, similar studies have been made of Brown Swiss cattle in Switzerland,[6] Telemark cattle in Norway,[1] Landrace swine in Denmark,[5] and Poland-China swine in the United States.[4]

In the present study two samples were taken, one of animals born in 1925, the other of animals born in 1935. Each sample consisted of 400 animals, 200 of each sex. A trend over a longer period could not be studied, because much of the foundation stock was so recently introduced to this country. The study could not be carried back of the imported ancestors because only the male line of descent is given in the English flock books.

The average inbreeding and *inter se* relationship coefficients are shown in Table I. The base dates are the average birth dates of the foundation animals. About 1.4 per cent of the heterozygosis which was present in 1911 had been lost by 1925. Similarly those born in 1935 had lost about 2.9 per cent of the heterozygosis that was present in 1914. There is no way of knowing how heterozygous the foundation stock was. The loss of heterozygosis per generation is much the same as that found in similar studies of other breeds of livestock. In the Rambouillet breed, the only other breed of sheep studied,[3] the total amount of inbreeding was higher, but the base dates were earlier (largely because Rambouillet importations practically ceased in 1906) and the rate per generation was a little lower than was found here.

Inter se Relationship

The *inter se* relationship coefficient is an estimate of the relationship existing between animals chosen at random from



DISTANT ANCESTORS OF THE HAMPSHIRE

Figure 18

Artist's sketch of the Old Wiltshire sheep, one of the breeds from which the Hampshires sprung. (From Low's *Domestic Animals of Great Britain.*)

the breed. It indicates how extensive and intertwined is the background of common ancestry. If it could be traced back to the time when the breed really first diverged from other breeds, it would express the distinctness of this breed from other breeds, except that it would not include breed differences caused by selection having been toward different goals in different breeds. Where animals of the same breed are all related to each other, a certain amount of inbreeding of course would result merely because of the continued pure breeding, even if mating were truly random within the limits of that breed. Under such circumstances the inbreeding (F) expected purely because of the average *inter se* relationship (R) would be $F = R/2\text{-}R$.

Actually the *inter se* relationships found were zero in 1925 and 0.5 per cent in 1935 which is much too little to explain the inbreeding found. Apparently a little separation into families has taken place and the breed has not developed as an absolutely homogeneous unit. Probably such separation as has taken place has been on a geographic basis rather than a deliberate attempt to keep families from interbreeding with each other.

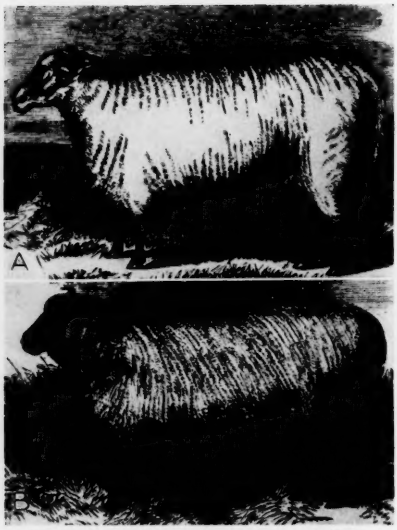
Prominent Animals of the Breed

The system of pedigree sampling used in this study made it possible to measure impartially the importance of various ancestors to the breed. The number of lines in which an ancestor was found, divided by the maximum number (800 in each sample) in which it could have ap-

TABLE I. Inbreeding and Inter se Relationship Coefficients.

Year	Base date of sample	Inbreeding		Inter se relationship
		Total	Per generation	
1925	1911	1.4 ± .4*	0.7	0.0
1935	1914	2.9 ± .6	0.9	0.5

* The figure following the ± sign is the standard error.



TWO EARLY HAMPSHIRE SHEEP
Figure 19

Artists' conceptions of two animals of the Hampshire breed after the Southdown outcross. The drawings were made about 1880 of a ram owned by Mr. Humphries (*A*) and a ewe owned by Mr. Rawlence (*B*). Humphries and Rawlence were two important early improvers of the breed. (From Spooner's *The Sheep*.)

peared, gives the direct relationship of the ancestor to the breed. An animal may also be related to the breed through its collateral relatives, i.e., brothers, cousins, etc., as well as through its own sons and daughters. Only two animals were related to the whole breed as much as two per cent. The first of these was Blendworth Herriad, No. 45215 (Figure 20*A*), who was bred in England but imported and used extensively by the Mt. Haggin Land and Livestock Company at Anaconda, Montana. His relationship to the breed in 1935 was 2.4 per cent. The other was Blendworth Herriad's son, No. C-1659 (Figure 20*B*), who had a relationship of 3.0 per cent to the breed in 1935. C-1659 was champion ram at the International Livestock Show at Chicago in 1926, as a lamb. He was used extensively as a sire and many prizes were won by his get, including four championships and five reserve championships at the International.

Interval Between Generations

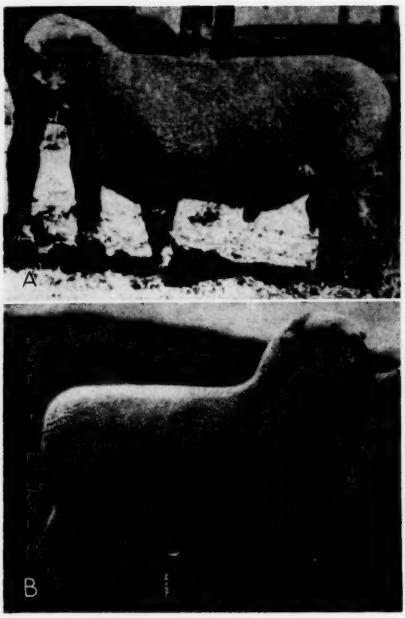
The interval between generations represents the average age of the parent when the offspring is born. The generation interval was longer in the 1935 sample (3.8 years) than in the 1925 sample (3.5 years). In a breed which is rapidly expanding in numbers a shorter generation interval is to be expected than in a breed relatively stable in numbers. This may explain the longer interval in 1935 when equilibrium in numbers had been approached. A slightly longer interval between generations, 4.2 years, was found by Dickson and Lush[3] in the Rambouillet breed than in either sample of the Hampshire breed. Possibly this results from the Rambouillet breed having approached a numerical equilibrium in this country at an earlier date than the Hampshire did.

Discussion

The period of very rapid expansion in numbers (1906-1912) based in large part on importations had its effect on the breeding system followed. The principal interest was placed on whether or not a sheep was purebred. Later, when the population numbers became more stabilized, more careful selection and increased attention to quality was possible.

The most plausible explanation for the apparent lack of balance between the observed inbreeding and *inter se* relationship coefficients is partial geographic isolation of various parts of the breed, rather than intentional inbreeding. A few breeders may have been following some definite linebreeding plan, but if so they must have been rare or the linebreeding must have been very mild. Sampling errors can hardly explain the entire discrepancy.

No one animal or family has ever dominated the Hampshire breed. This is particularly striking when compared with the Shorthorn breed which was 55 per cent related to Favourite and 45 per cent related to Champion of England in 1920. This lack of prominent animals is perhaps due to the relatively recent introduction of the breed to the United



PROMINENT HAMPSHIRES
Figure 20

Only Blendworth Herriad (*A*) and his son, C-1659 (*B*) showed more than 2 per cent relationship to the breed.

States, as well as to the large numbers in those importations. Several practices that are prevalent among sheep breeders tend to emphasize the flock rather than the individual animal. In England the system of flock registration has this effect. In the United States all animals are registered individually but are given flock numbers instead of names. Most of the breed promotion work, publicity, and announcements of show-yard winnings emphasize the owner's name rather than the individual sheep.

Whether Blendworth Herriad or his son, C-1659, or yet some other prominent animal is to become as important in the pedigrees of American Hampshires as Anxiety 4th and Don Carlos and Beau Brummel are in the pedigrees of American Herefords or Favourite and Champion of England are in Shorthorns will depend not only on their own inherent merit but also on whether a strain closely related to them is preserved. Unless some policy of linebreeding to the prominent ancestor is followed, his relationship to his descendants will be reduced by half with each succeeding generation. With a breed as large numerically as the Hampshire, an animal's relationship to the whole breed could thus become very small before even extensive use of his descendants would spread his inheritance all through the breed to the extent that it could be diluted no further.

Summary

1. The inbreeding which occurred in the Hampshire breed of sheep since it was brought to the United States was 1.4 per cent in 1925 and 2.9 per cent in 1935. About 0.7 to 0.9 per cent of the existing heterozygosis was lost per generation during the period studied.

2. The *inter se* relationship found was zero in 1925 and 0.5 per cent in 1935. These figures indicate that some separation into families has taken place.

3. Only two animals had a relationship to the breed of more than two per cent.

4. The average interval between generations was 3.5 and 3.8 years in the two samples.

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INHERITANCE OF A WHITE FORELOCK*

In a Mulatto Family

ADELAIDE M. CROMWELL

THERE is in the literature a number of conflicting theories regarding the origin of piebald spotting in man. According to Karl Pearson, a pure race crossing with a mixed race results in a hybrid between the races with markedly different degrees of pigmentation.[7] In such a population Pearson believed that piebalds are likely to appear "de novo." Pearson also is of the opinion that piebaldism is neither a Mendelian unit character nor a latent unit character because there is no evidence of its previous occurrence in the pure races whose crossing leads to the piebalds. Many piebald pedigrees show that sufficient cause for piebaldism exists when a pure red or white is crossed with a pure dark race and the resulting hybrid is mated with either pure race. Indeed the light race itself may be merely an albino variety of the dark race, for in several cases of dark-race pedigrees, piebalds are found occurring in stocks where albinism has also occurred. When a black dog is crossed with a pure albino of another race, the F_1 is never a true piebald, but if the F_1 is crossed with an albino, black and white piebalds will be among the offspring.[7] Much the same changes seem to arise from like crosses not only in mice and axolotl but also in man.

Miller holds that albinism itself may appear in almost any degree, from a mere colorless spot to a complete lack of pigment in the hair, skin, and eyes.[5] Rizzoli published in 1877 a record showing a forelock of white hair running through six generations of a family comprising 490 individuals in the direct line of descent. A similar account by Harriman appeared in 1909. In this case, however, the "flare" cropped out in six successive generations of a family numbering 138 members in the direct line. Other colorless patches are said to be found on various parts of the body in this family. Miller concludes by saying that the lock crops out only in those families one parent of which is thus marked, and, contrary to Pearson's idea, that it behaves as a simple dominant with normality as recessive. In addition, Miller says it follows closely the Mendelian law. He presents two types of matings possible to produce this effect: DR $\times$ RR and DR $\times$ DR. According to this supposition one should expect to find the leucotic spot in one-half of the children in families where one parent is marked and never in children of unmarked parents.

Persons showing a much less extensive area affected, often only a single patch, have been conveniently called spotlings. In piebalds the belly and chest are involved and often associated with a flare on the scalp and "garter" or "stocking" markings. In spotlings this arrangement and extent may be wanting. Piebalds and spotlings are cases of partial albinism. In natives of Nyasaland observed by Staunus, the hairs were devoid of all pigment.[9] He concluded that piebalds were rare, spotlings less rare, but albinism common.

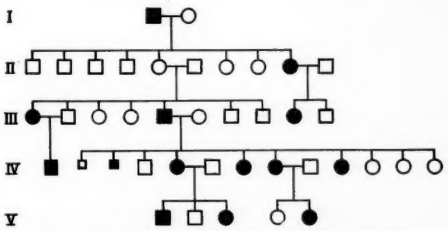
Cockayne offers an additional bit of information when he says that piebalds are uncommon in both white and Negro races but far less observable in white.[2] He implies, however, that they do appear in white races. This fact contradicts the theory, referred to above, that piebaldism comes from race crossing. Cockayne also says that piebaldism can affect either sex and does not act numerically as a pure dominant. In some of his cases he found the eyelashes to be alternately white and brown and in other cases the eyebrows were similarly affected.

Even in Barton's Case as presented by Pearson, the affected produce affected, and he believes the characteristic not

*Contributions from the Department of Zoology, Smith College, No. 189.

to be recessive. However, in this case he agrees with Cockayne in saying that none of the affected can be looked upon as a pure dominant.[8] He found no case in which an apparent normal marrying a normal transmitted the character.

Gates suggests that spotting be considered as arising, in certain cases, as a somatic segregation, repulsion, or dilution effect and not as due to an inherited invisible spotting factor.[2] Once this spotting condition has arisen, it becomes fairly stable. Because the condition becomes stabilized in the germ plasm, Gates prefers to regard it as a mutation following crossing. In man, spotting often follows a characteristic pattern beginning as a blaze on the forehead, with spotting of the arms, the chest, and the lower extremities. Gates says, however, that the sporadic manner in which spotting appears remains to be accounted for. The genetic facts connected with its inheritance do not seem to fit in with any established genetic theory. Gates' data indicate that the peculiarity of the white forelock is inherited as a simple Mendelian dominant, chance accounting for any observable absence of affected males. This feature resembles the white "star" markings seen on the foreheads of horses and cattle and frequently accompanied by a white patch on the extremities. Gates, too, says that unaffected members never transmit the flare; therefore it cannot be a regular recessive. If the character is a simple dominant, which it is not, the small numbers showing the flare might be accounted for in the assumption of the decreased viability of such heterozygous individuals. In the case of Holmes and Scofield (1917), heterozygous females do not show the mark. Therefore it appears dominant in the males and recessive in the females. Spotted mulattoes arise from an ancestry in which different types of skin pigmentation have been involved. The fact that spotting appears rarely and sporadically among the innumerable mulatto crosses tells strongly against its being the result of an inherited spotting factor. It arises as a mutant in the hybrid stock, the original spotted individual being heterozygous and the



PIEBALDS IN FIVE GENERATIONS

Figure 21

Pedigrees of a mulatto family showing the inheritance of a white forelock. The gene was introduced into the family by a white slaveholder, I-1, who had children by a dark mulatto, I-2. Although II-5 did not have a white forelock, she transmitted it to her children, indicating that in this instance the trait was irregular in dominance. The small symbols indicate death in youth.

piebald condition dominant to normal mulatto pigmentation.

In the particular case I have studied no attempt is being made to disprove any of the theories of the above mentioned authorities, but all statements made are thoroughly confirmed. I have purposely left some things out because I was not sure of the validity of the information. The family studied had its beginning, so far as I was able to trace, with a white German, named Blooker Hansborough, who was a slave holder in Culpepper County, Virginia. Blooker, whose real name was probably Bluecher, had, as was the custom, two or three concubines by whom he had many sets of children. I am tracing the history of one set only, although enough information might be obtained to trace another branch with which I am acquainted.

Blooker Hansborough was white, and he had the white forelock. It is not known whether he was or was not spotted. He had children by one Lucinda Wormley, who was quite dark and apparently normal. These two had eight children—four boys and four girls—all of whom were without the forelock and any markings except one girl, Mary Hansborough. As is shown on the accompanying chart, Mary married an apparently normal man and had two children, a girl with the forelock and a boy

without it. The girl, Marian Cook, has no children herself, but she is of particular interest because she has an extremely wide forelock, and her patch extends very noticeably down on her forehead, almost to her eyebrows. Her brother has no children either. All the seven normal children, except one, have had offspring who have not had the forelock.

This other one is the most interesting of all. She was Rose Hansborough and was apparently normal. The truth of this is avowed by her son from whom I have the evidence. As Rose had raven hair there would have been no mistaking the patch if it had been present. Rose was in no way spotted on the body. She married a normal man, Warner McGuinn, and by him she had six children, three girls and three boys, one of either sex being marked by the presence of the forelock. The girl, Sarah McGuinn (Becks), married a normal man, but they had a son who had the streak. All of their normal children had normal offspring. The son with the forelock, John McGuinn, however, married a normal woman and by her had ten children, three boys and seven girls. Of these ten children five were marked by the forelock, one boy and four girls. The boy died in infancy. Two of these girls have married, and of the three children of one of the girls two children have the forelock; while of the two children of the other one child has the forelock. A complete history of the presence or absence of the forelock and spotting appears in the pedigree chart, Figure 21.

Discussion

There are a few quite interesting things about this particular case. In the first place, the trait was introduced into the pedigree by Blooker Hansborough who was pure white. He married a dark woman, but not a pure black. All of the other marriages in this pedigree were of mulattoes. There were no consanguineous marriages. This is contrary to the statement of Pearson that the forelock appears only after race crossing but agrees with Cockayne who has noted it in white races.

The second unique point is that Rose Hansborough, a daughter of Blooker and Lucinda, did not herself have the forelock, but carried the gene or genes for it because she passed it on to two of her offspring. In no other pedigree history of the inheritance of the forelock, in so far as I am able to ascertain, has this been noted. Therefore this trait is neither dominant nor must it originate from race mixture. Indeed, although the authorities disagree in the actual potency of the gene or genes in inheritance, they do agree on the fact that it has never been observed in the offspring of parents of whom one, at least, does not have the trait. Such was certainly the case in my study.

The inheritance of the white forelock is not that of pure recessiveness, nor does it follow exactly the rules of Mendelian inheritance. With the exception of the original family the inheritance seems to follow more closely the three to one ratio, and the results of the first mating might well have been due to chance. If such were true, the trait would appear to be more of a recessive than of a dominant nature, but because the trait appears more often than a recessive should appear and less often than a dominant, no definite rule of inheritance can be laid down for it. It is indisputable that it is inherited and that it can and does arise from pure races. In this instance it was shown to skip a generation.

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